



This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

### Usage guidelines

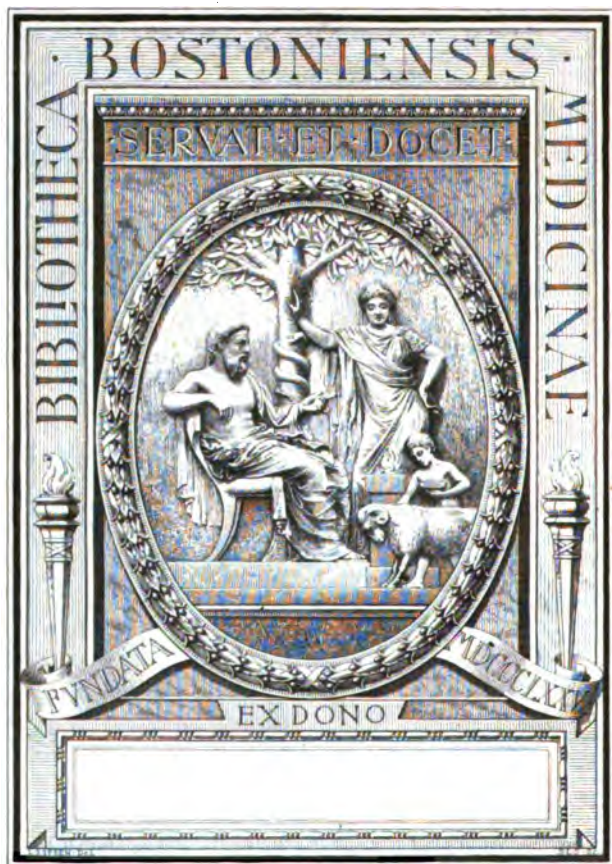
Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

- + *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.
- + *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.
- + *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.
- + *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

### About Google Book Search

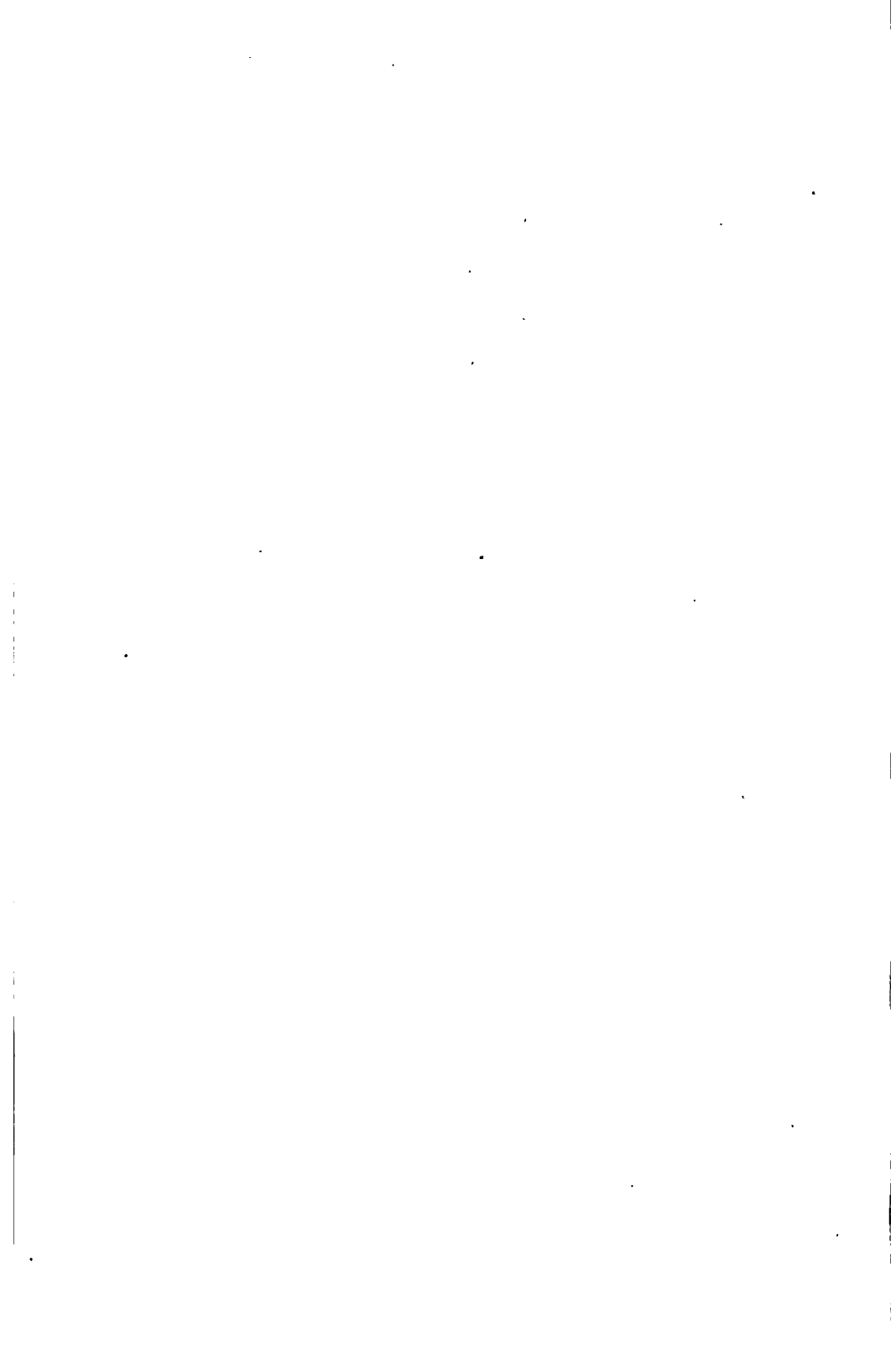
Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at <http://books.google.com/>













REVIEW  
OF  
NEUROLOGY AND PSYCHIATRY

EDITOR  
ALEXANDER BRUCE  
M.D., F.R.C.P.E., F.R.S.E.

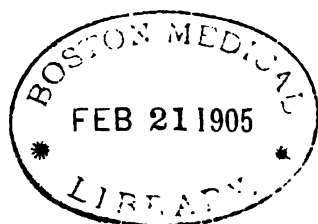
ASSISTANT EDITOR  
EDWIN BRAMWELL  
M.B., F.R.C.P.E., M.R.C.P.LOND.

VOLUME II.



EDINBURGH  
OTTO SCHULZE & COMPANY  
20 SOUTH FREDERICK STREET  
1904





**EDINBURGH**  
*Printed by*  
**TURNBULL & SPEARS**



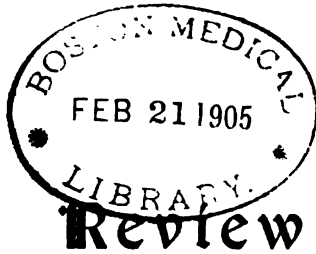
# Contents

## ORIGINAL ARTICLES.

	PAGE
A MORPHOLOGICAL CONTINUITY OF GERM-CELLS AS THE BASIS OF HEREDITY AND VARIATION. By J. Beard, D.Sc. . . . .	1, 114, 186
A CASE OF INTENSE AND GENERAL MUSCULAR ATROPHY CAUSED BY ENCEPHALOMYELITIS. By William G. Spiller, M.D. . . . .	34
LUMBAR PUNCTURE. By C. Macfie Campbell, M.B., Ch.B. . . . .	39
A CASE OF PRIMARY SCLEROSIS OF THE POSTERIOR COLUMNS, FOLLOWED BY DISSEMINATED SOFTENING OF THE OTHER WHITE COLUMNS. A Contribution to the Study of the Subacute Combined Degenerations of the Spinal Cord. By Joseph Collins . . . . .	97
CASE OF PNEUMOCOCCAL MENINGITIS AND SOME RECORDS OF THE VALUE OF THE CYTOLOGICAL EXAMINATION IN CASES OF MENINGITIS. By W. B. Warrington, M.D., M.R.C.P. . . . .	107
SOME UNUSUAL CASES OF TABES DORSALIS (with Remarks). By Ernest S. Reynolds, M.B., F.R.C.P. . . . .	177
THE STATE OF THE EYE MUSCLES IN ORGANIC HEMIPLEGIA. By S. A. Kinnier Wilson, M.A., M.B., B.Sc. . . . .	265
VARIATION IN ITS RELATION TO THE ORIGIN OF INSANITY AND THE ALLIED NEUROSES, being the Morrison Lectures for 1904. By John Macpherson, M.D., F.R.C.P.E. . . . .	285
THE DIAGNOSTIC VALUE OF THE PLANTAR REFLEX. By Stanley Barnes, M.D., B.Sc., M.R.C.P. (Lond.), M.B., Ch.B. (Birm.) . . . .	345
A PRELIMINARY NOTE ON AN ABERRANT CIRCUMOLIVARY BUNDLE SPRINGING FROM THE LEFT PYRAMIDAL TRACT. By G. Elliot Smith, M.D. . . . .	377
HEREDITARY SYPHILITIC TABES (Juvenile Tabes). By R. T. Williamson, M.D., F.R.C.P. . . . .	425
A CASE OF SENILE COMBINED SCLEROSIS OF THE SPINAL CORD. By S. A. Kinnier Wilson, M.B., B.Sc., and Dr O. Crouzon . . . . .	431

	PAGE
DISSEMINATED SCLEROSIS. An Account of the Microscopical Examination of Three Cases, with some Observations on the Pathogenesis of the Disease. By A. F. Tredgold, L.R.C.P. (Lond.), M.R.C.S. (Eng.) . . . . .	497
A METHOD OF PRODUCING A SELECTIVE DEPOSIT OF SILVER IN BLOCKS OF TISSUE, AND OF OBTAINING SECTIONS SUITABLE FOR TONING WITH GOLD, PLATINUM, AND OTHER METALS. By W. Ford Robertson, M.D. . . . .	522
OPTIC NEURITIS IN CASES OF INTRACRANIAL TUMOUR, WITH SPECIAL REFERENCE TO THE NEUROGLIAL CHANGES PRESENT. By Robert A. Fleming, M.D., F.R.C.P.E. . . . .	569
FUNCTIONAL ASTEREOGNOSIS. By George E. Rennie, M.D., M.R.C.P. (Lond.) . . . . .	579
ON THE SEGMENTATION OF THE INTERMEDIO-LATERAL TRACT. Preliminary Communication. By Alexander Bruce, M.D., F.R.C.P.E., and C. Macfie Campbell, M.D. . . . .	582
NEUROMERES OF THE RHOMBENCEPHALON OF THE PIG. By O. Charnock Bradley, M.B., F.R.S.E. . . . .	625
A FURTHER STUDY ON THE SENSORY SEGMENTAL ZONE OF THE UMBILICUS. By William G. Spiller, M.D., and T. H. Weisenburg, M.D. . . . .	681
A CASE OF INTRA-CRANIAL ANEURISM IN A YOUNG SUBJECT. By Alexander Bruce, M.D., F.R.C.P.E., and W. B. Drummond, M.B., C.M., F.R.C.P.E. . . . .	737
THE MODE OF TURNING, IN WALKING, OF ORGANIC HEMIPLEGICS AND SPASTIC PARAPLEGICS. By Leonard J. Kidd, M.D. . . . .	742
SALT STARVATION IN THE TREATMENT OF EPILEPSY. By William Aldren Turner, M.D., F.R.C.P. . . . .	793
VERTICAL EYE-MOVEMENT TESTING IN THE POSITIONS OF ABDUCTION AND ADDUCTION. By Leonard J. Kidd, M.D. . . . .	799

8771



of  
**Neurology and Psychiatry**

**Original Articles**

**A MORPHOLOGICAL CONTINUITY OF GERM-CELLS AS  
THE BASIS OF HEREDITY AND VARIATION.**

By J. BEARD, D.Sc.,  
University Lecturer in Comparative Embryology, Edinburgh.

*"Die Folgerungen aus dieser Lehre von der Continuität der Geschlechtszellen sind fast unabsehbar für die gesamte Biologie."*—W. WALDEYER.

PREFACE.

IN the following pages it is proposed to give an account of observations and conclusions, relating to what in their origin, at any rate, were purely embryological problems. The researches, commenced in the autumn of 1888, have now extended over more than fifteen years, and they were undertaken, and for many years continued, solely with a view of determining by observation the apparently simple question of the mode of development of a vertebrate animal. They had not proceeded very far before it came to be recognised that whether or not a "direct development"<sup>1</sup> was possible, that presently carried out in the higher animals had every appearance of being anything

<sup>1</sup> Under the still prevailing view of "direct development" the hen, for example, produces from the tissues of her body new eggs, while it is the immediate task of the fertilised egg to give rise to a new hen. In these few simple words are contained, as will be seen anon, three of the most serious errors, epigenesis, direct development, and somatic origin of germ-cells, into which it was ever the misfortune of any branch of science to fall.

but direct; indeed, of being based in an antithetic alternation of generations.<sup>1</sup> This was of such a kind that the fertilised egg gave rise to an asexual foundation or larva, termed by me the phorozoon or bearing-animal, and that in some way or other upon the latter there arose an organism, termed the embryo, which by reason of its endowment with sexual organs may be described as the sexual form of generation.

To the writer the truth of this was convincing enough ten years ago, but at that time the state of the investigations was not such as to win over other scientific workers—with a single exception!—to the opinion that there was the slightest reason for doubting any of the fundamental tenets of embryology then current. But now it is quite certain that the phorozoon and alternation of generations have come to make a permanent stay with the science of embryology, and that its votaries can as little dispense with them as can Nature in her workings.

Since the standpoint was attained more than ten years ago, no facts adverse to it have been encountered. From then till now the course of research has proceeded straight as an arrow to the climax. The rebuffs, which have had to be faced and borne, have not come from the investigation itself: it has been quite free from check, except that which at any time lack of material may impose upon the worker. The doctrine has never been seriously attacked: in all these years it has simply been ignored. It is rapidly gaining supporters now, but for long it did not win many—the truth never does at first! For myself I have been content to follow out the inquiry, and from time to time, as opportunity offered, to glean a few more facts supporting this theory of development. During part of this period a watch was kept for something equivalent to the formation of spore-mother-cells in the higher plants or Metaphyta, but in vain. In the earlier years, as at length apparent, the search had not been made in the right place.

The investigator is often the creature of circumstances. These in the present case brought about a research into the early history of the germ-cells without at the start associating with this inquiry any ideas concerning spore-mother-cell-forma-

<sup>1</sup> A botanical term. Where in a life-cycle there are two organisms differing in their organs and in their total organization, and when there is thus no homology between them or their parts, the alternation of generations is said to be "antithetic."



tion or alternation of generations. Only when this work had reached a preliminary finish, and when the results had been drawn up in diagrammatic form for purposes of publication, did the full force of the facts discovered become evident.

As now fully recognised, the more recent portion of the work—that upon the germ-cells and their problems—is an absolute necessity for the formulation of any clear and scientific conception of the nature and mode of development of the higher animals. Its results have not only confirmed the conclusions of former years, but they have served very considerably to extend these. The germ-cell researches, including those into the problem of the determination of sex, have established antithetic alternation of generations as the mode of the development.

Of the consequences that follow out of the researches it is, perhaps, not too much to say that they are immeasurable. Looking back now at the starting-point, it must be admitted that the track has been long and lonely. The lonesomeness of research along a new and untrodden field is, indeed, as the solitude of the pathless desert !

Among the pioneers in this region the name of Waldeyer is, perhaps, the most prominent. And there is a fitness in prefacing the present writing with a passage quite recently written by this veteran observer. As will appear in the sequel, the proof of a morphological continuity of germ-cells from generation to generation was first really brought by the writer in his published researches. In them was afforded for the first time the possibility of following in a distinct and tangible form, and without the assumption of the continuity of an hypothetical intangible germplasm (Weismann), the track of germinal continuity from one generation to the next. Waldeyer writes: "The consequences of this doctrine of the morphological continuity of sexual cells are almost unbounded for every branch of biology." No earnest investigator can ignore the immense, the overwhelming importance of this continuity for the science of embryology. It and the various facts associated with it are bound sooner or later to revolutionize completely the ideas and conceptions of zoologists, anatomists, and embryologists. They will relegate the dogmas of epigenesis and direct development to the list—a pretty long one already—of former erroneous doctrines of science, and they will open up new and important

pathways of research and knowledge, of which at the moment no conception whatever can be formed. In other directions the doctrine and its allied facts will doubtless prove themselves to be equally fruitful of results. Without the least doubt they form the basis of that portion of pathology treating of the tumours, and the writer is informed that their bearings upon the problems of insanity are simply incalculable. It is this assurance that accounts for, and may excuse, the appearance of an embryological document in the pages of this journal.

#### INTRODUCTION.

The main objects in view in the following pages are the problems of heredity and variation, and the light thrown upon these by the results of a study of the life-cycle of development and of the important part played by the germ-cells in this. It is out of question, it is, perhaps, not needful, to give an account in detail of all the researches, or of the work of other observers, whose results may be required for the elucidation of some point or other. The present writing, therefore, will be little more than an epitome of results and conclusions, and for fuller information reference must be made to the original memoirs.

The problem of heredity is almost the greatest one in embryological science.<sup>1</sup> Owing mainly to the writings of Brooks, de Vries, O. Hertwig, Naegeli, Herbert Spencer, and above all others, Galton and Weismann, heredity and its problems have occupied a prominent position in the discussions of recent years. The progress of research into the life-history of the cell, the structure and functions of the nucleus, the phenomena of cell-division, more especially those attending the "ripening" of the "sexual products," have naturally played important parts in these. Indeed, so much has this been the case, that H. F. Osborn might well say the prophetic words, "the study of heredity will ultimately centre around the structure and functions of the germ-cells." This is hardly the place for a full history of these discussions and theories; what

<sup>1</sup> "These phenomena (of heredity) constitute the basis of all evolutionary science and the very central problem of natural history."—W. BATESON, in "Mendel's Principles of Heredity," Cambridge, 1902, p. 1.

is proposed is to indicate the broad and obvious bearings of certain of my results, relating to the history of the germ-cells and the course of the life-cycle, upon the general problems of heredity and variation.

One may dismiss the older ideas upon the subject, popular and scientific, and take as the starting-point Galton's "stirp" (1875). This was conceived of as something left over from every development, a material basis, from which ultimately the next generation took its origin. In the conception of this "stirp" we have for the first time a recognition, dim and indistinct though it be, of a tangible something, connected in some way with the germ-cells, out of which the phenomena of heredity proceeded. A much larger space in the history of heredity is occupied by Weismann's theory of the germplasm. His doctrines have been elaborated and extended with infinite pains and untiring research during the last twenty years.<sup>1</sup> In the lectures referred to below, in the English editions of his essays, and in the "Germplasm," published in 1893, full accounts of his views may be found. I have no space here for an adequate statement of them. The whole doctrine is based in an hypothetical germplasm of great complexity and endowed with enormous potentialities. It is not so much in a morphological continuity of germ-cells that Weismann's conclusions are founded, as in a continuity of a germplasm which may even be in somatic cells. More than once he has expressly stated, and certainly he has never retracted it, that "in some cases at any rate," germ-cells may arise from somatic cells. And it is only for the group of the Insecta (1) that a real morphological continuity of germ-cells is admitted by him, while for all other Metazoa the theory advocates the continuity of an hypothetical germplasm alone.

*Any theory of heredity to be successful must have its basis in the facts and mode of Metazoan development.* The great weakness of Weismann's theory of the germplasm is that it is not in accord with the facts of development, and not based in these. And while, on the one hand, since the appearance of my results upon the germ-cells of *Raja* in preliminary form in 1900,

<sup>1</sup> Weismann's address, "Ueber die Vererbung," was published in 1883, while his latest work upon heredity, "Vortraege ueber Descendenztheorie," in two volumes of some 900 pages, appeared in 1902.

Weismann does not really now deny a morphological continuity of germ-cells—as he previously did when face to face with Nussbaum's conclusion (for which see later)—he practically never really admits it<sup>1</sup>—certainly not in my sense. The existing morphological continuity is replaced by that of an hypothetical substance, the germplasm, whose necessity I cannot recognise. Moreover, as an upholder of the recapitulation-theory and of direct development, Weismann's conception of the course of the development from one generation to the next differs *in toto* from that which my researches have led me to adopt as the only true and sole possible one.

It has been urged, as, indeed, might have been anticipated, that the writer's conclusions were but slight variations upon the earlier ones of Weismann. Barring the acceptance of the biophores, which as pangenes were originally conceptions of de Vries, almost the only other real resemblance between Weismann's conclusions and mine is our agreement as to the impossibility of epigenesis, and as to the truth of evolution or unfolding. It is, no doubt, quite true that the hypothesis of the germplasm and its continuity offer explanations of any number of things, almost identical with those given under a morphological continuity of germ-cells. Again and again in Weismann's writings were one to substitute the expression "germ-cell or germ-cells" for "germplasm," facts or probabilities would be stated. But everywhere the actual thing named is germplasm, not germ-cell! And quite apart from the difficulties associated with the conception of the germplasm and its continuity, the assumption of its existence is now unnecessary. And it must not be forgotten that Weismann's views and conclusions admit, and are founded in, direct development in the Metazoa, recapitulation in development, and "in some cases at any rate," somatic origin of germ-cells, all of which are under my conclusions emphatically denied.

In order to obtain a clear insight into the process or processes by which, in a wide sense, germinal continuity, resulting in the phenomena of heredity, is brought to pass, it is a requisite

<sup>1</sup> Except for the Insecta. But of what value is the admission of this exception? If this be the only group in which there be a real morphological continuity, the assumption of a germplasm may be needed for the explanation of other cases. But if there be reason to believe in the general occurrence of a morphological continuity of germ-cells, the assumption of the existence of a germplasm becomes an unnecessary multiplication of causes, and as such unscientific.

postulate that an uninterrupted and continuous panorama of the whole course of development from one generation to the next should be secured. Heredity must be dependent upon some sort of germinal continuity; whether of a special germplasm in Weismann's sense, or a consequence of an uninterrupted sequence of germ-cells, or a result of an intracellular pangenesis, or something else.

In this way it comes to be a problem of embryology and development, and as such it falls within the province of the embryologist. This being so, is it not remarkable that the chain of germinal continuity should hitherto not have been grasped completely in any single case? From my researches on the germ-cells, it is clear that hitherto no complete survey of the development from one generation to the next has ever really been made. One phenomenon in the Metazoan life-cycle has entirely eluded the observation of embryologists; or, if they have noted or recorded it, they have failed to realise its full significance. This is the formation of the primary germ-cells, with the epoch at which these appear upon the scene.

Their very early origin—before any trace of an embryo had been laid down—was long ago recorded in certain cases, among others by Weismann, Buetschli, Grobben, Ritter, Metschnikoff, and O. Hertwig. But these very instances only serve to strengthen my contention, for in them the few (primary) germ-cells—from two to eight in number—were apparently so insignificant that their formation at a particular time seemed to be an incident of no moment; and its discovery, like many other important finds, was passed over, because no estimate could be set upon its value.

#### I. HISTORICAL CONCERNING THE GERM-CELLS.

The early origin of germ-cells in a few cases, *Moina*, *Cecidomyia*, *Chironomus*, and *Sagitta*, has been common knowledge for a long time. But from these cases, or, indeed, from the later ones, described by Nussbaum and Eigenmann, none ever dreamt either of setting up a new conception of the course of (Metazoan) development, or—and this is very important—of advocating a morphological continuity of germ-cells from generation to generation. If the germ-cells be not somatic in origin,



the mode of development cannot be that assumed—without proof—by investigators for the past hundred years! And, as for a morphological continuity, the most that any zoologist ever ventured is, indeed, represented in the following words of Weismann, written in his latest work (2): “Could we assume that the egg on commencing development at once divided into two cells, of which the one gave rise to the entire body (soma), while from the other only the germ-cells contained within this arose, the matter would be theoretically very simple; we should say that the germplasm of the egg doubled itself, as happens to the nuclear substance in every cell-division, and that this divided into two equal halves,” one of which formed the body of the individual, whilst the remainder, its germplasm remaining latent, only manifested activities sufficient to give rise to and to impress its stamp upon certain cells, its products, which became the germ-cells. He proceeds to say that this has as yet only been proved to be the case in the dipterous insects. But even in this instance it may be questioned whether Weismann’s account is sufficiently precise. For not even here is there witnessed a division of the egg into two halves, somatic and germinal respectively in destination. In fact, nowhere, so far as is known, does the ancestral or primitive germ-cell arise as early as the first division. And, when Weismann speaks of its origin “after the first couple of divisions,” this is merely an euphemism; for, of course, he is well enough aware that the number of the divisions of the fertilised egg, prior to the appearance of the primitive germ-cell, is invariably more than two, and very often four.<sup>1</sup> That is to say, except in mammals, where apparently it may appear at the third mitosis, almost the earliest period, if not the earliest, is the fifth cleavage phase, as in *Cyclops*, and possibly *Ascaris* and *Strongylus*, and it may appear first at about the tenth division, as in *Scyllium* and *Raja*.

In the work under reference, passing from the insects,

<sup>1</sup> Concerning the epoch at which the primitive germ-cell and the primary germ-cells appear in the Diptera, etc., there are no precise statements in the literature, or in Weismann’s published researches. In no instance would they appear to arise earlier than in *Nereis*, i.e. with the fifth cleavage. Possibly, though not certainly, in mammals (*Tupaia*, *Tarsius*) the primitive germ-cell may be a product of the third cleavage. This appears to be the explanation of Hubrecht’s finds of a sac of seven cells with one clear cell in the centre, out of which cell alone the whole embryo takes its birth, according to Hubrecht.

Weismann proceeds to give an account of the origin of the sexual cells in the Hydrozoa, or hydroid polypes. These marvellous researches, published in 1883, apart from the valuable facts they have added to our knowledge, have unfortunately served to retard it in certain directions. They have been held to have established a somatic origin of germ-cells, and they have been, and still are, used by Weismann to show that for a very prolonged period, even for scores of cell-generations, the "germinal track" of Weismann may lie in somatic cells.

In the foregoing passages there are implied two fundamental errors, which have retarded our knowledge of germinal continuity and how this is effected, and which have delayed a recognition of the true course of the life-cycle. It is assumed to be the task of the fertilised egg to give rise straightway to a new "embryo," and the hydroid polype has been—and, *mirabile dictu*, still is!!!—regarded as morphologically the equivalent of the "embryo" or sexual form of any of the higher animals. It has been ignored, that in all the latter, from the worms to man himself, the products of the early cleavage are concerned, not in the formation of an embryo, but of an asexual foundation or larva. Regarding the Hydrozoa, it has not been clearly recognised that in origin, mode of growth, and other peculiarities, the hydroid polype corresponds to the sporophyte of plants, that it is not the bearer of sexual products, the forerunners of these arising from certain apical cells, and it has been wrongly believed that the medusa or its equivalent is homologous with the hydroid polype. To prove that in the Hydrozoa germ-cells are somatic in origin, it must be demonstrated that they have their birth from some tissue or other of the sexual generation, the medusa. It need hardly be remarked that Weismann's own researches amply disprove this.

In 1880 Nussbaum enunciated the conclusion, which he arrived at from the appearance and size of the germ-cells of the embryo frog, that these must be special cells of the egg-cleavage, which at no time took a share in forming the individual, but which were intended for future generations. Unfortunately, he has never by research followed up the matter to its end. I have already elsewhere described this as a "brilliant idea," and as one result of my researches, but one only, I have emphatically endorsed it. A like conclusion was enunciated in 1892 by

Eigenmann for a Teleostean fish, *Micrometrus*, and his work contains many more observations upon the germ-cells than does that of Nussbaum. Thus, he recorded their presence far forward in the embryo, the disappearance of some of them, and their migrations, in addition to surmising that they dated back to the fifth cleavage-phase. Neither of these observers made any attempt to set up by observation a morphological continuity of germ-cells; for with neither did the mode of the development, the true life-cycle, lie within the scope of the observations. When, therefore, Nussbaum<sup>1</sup> in a recent lecture sets up a morphological continuity of a sort, this is based merely in an incorrect and loose dictum that the segmented egg divides into the somatic cells and the germ-cells. When cleavage is really finished, an epoch which is not the same for all animals, *there are no somatic cells present!* Neither Nussbaum's view of matters, nor Waldeyer's assumption of the division of the egg into two halves, somatic and germinal respectively, nor even Weismann's most recent statements, afford us any sound and real conception of the actual facts.

It is not sufficient for our purpose to study the first appearance of germ-cells in embryos—by so doing we may conclude their somatic origin, whereas, in reality, they exist prior to the appearance of any trace of a soma. *This is a fact of observation, and its existence removes the justification for any and every assumption.* It is illogical to conclude that the problem is solved in all its aspects, or in anything like all, when from the sizes and other characters of the earliest germ-cells in embryos we have referred them to the cleavage. To trace the whole chain of continuity, to reach the conception of a morphological continuity of germ-cells, we must be able to form an adequate picture to ourselves of all that happens to the products of the egg from the moment of fertilisation until the appearance of a new egg. This was done, and for the first time, in the various results of my investigations, published in the years from 1889 to 1902. And it is these researches, along with the little portions of the life-cycle filled in from the work of others (Boveri, E. B. Wilson, and

<sup>1</sup> Nussbaum, M., "Die Vererbung erworbener Eigenschaften," in *Sitzungsber. Niederrhein. Gesell.*, Bonn, Feb. 16, 1903, *loc. cit.* p. 2: "Es teilt sich das gefurchte Ei in die somatischen und in die Geschlechtszellen." It would be of interest to learn upon whose observations this statement rested—certainly not upon Nussbaum's work or mine!!!

Semper), which first really established for the Metazoa a morphological continuity of germ-cells. Such a continuity is nowhere shown in the researches of Nussbaum, or Boveri, or Weismann, or in those of any other observer. And how Prof. Waldeyer can believe this to have been the case, I can as little conceive, as I can imagine the continuity of an hypothetical germplasm to be the same thing as an actual morphological continuity of germ-cells.

Indeed, even as Weismann advanced the conception of a germplasm, he denied the validity of Nussbaum's conclusion mentioned above—and Nussbaum's idea was undoubtedly in the direction of a morphological continuity—and he decidedly rejected the view "because as a matter of fact the sexual cells of all plants and those of most animals do not separate themselves from the beginning from the somatic cells" (3).

And this is just the question at issue! The statement contains two fallacies, and these rob it of all force. Taking these in the order of their occurrence, the first is, that the sexual cells of all plants do not separate themselves from the beginning from the somatic cells. Probably all the higher plants, the Metaphyta, are here referred to, for in many of the lower plants all the cells might be regarded as potentially reproductive or "sexual." In the higher plants the "sexual cells" do appear at a very early period *in the sexual generation*. The higher one ascends the earlier is this epoch; for in the flowering plants, for instance, the life-span of the sexual generation, the gametophyte, is exceedingly short, and it is concerned solely with the differentiation of, and the provision for, the sexual cells. These latter certainly do not appear as such in the asexual generation or sporophyte, nor is it to be expected that they should. Were they to do so, the sporophyte would lose this character, and become a gametophyte. Moreover, even in the asexual generation, the sporophyte, the morphological continuity is unbroken, for in this the future germ-cells are represented by their direct ancestors, the one or more cells forming the apex.

Of this continuity in plants, Noll eloquently writes as follows: "The vital capacity of the cells of the functioning permanent tissue is always limited in time—mostly, indeed, very closely so. Without limit, on the contrary, and never finding a natural close, the vital power of the embryonic substance is pre-

served. This it is which forms the growing points of the perennial plants, and from this, as Sachs first demonstrated, the growing points of the sexual progeny are directly derived through the substance of the germ-cells. This embryonic substance does not age; it produces new passing individuals, but it is permanently preserved in their progeny: it is always productive, always growing young and increasing. Thousands upon thousands of generations which have arisen in the course of millions of years were its products, but it lives on, in the youngest generations with the power of giving origin to coming millions. The individual organism is transient, but its embryonic substance, which produces the mortal tissues, preserves itself imperishable, everlasting, and constant. Regarded from this standpoint, the differences in the duration of life between short and long-lived plants, between annual herbs and the thousands of years old giants of the plant race, appear in another light. Out of the embryonic substance of that lime-tree of Neustadt every year new leaves and buds form, but these remain in connection with the dying remains of structures of earlier years. In the annual plant, on the contrary, the embryonic substance separates itself every year in the embryo from the mortal remains, and forming new branches, leaves and roots, becomes a completely new individual. At the basis of the old and well-known dictum of Harvey, 'omne vivum ex ovo,' there thus already lay the continuity of the embryonic substance. This is, at the same time, in eternal youth and organic immortality the substance of the unicellular organisms, which, reproducing by fission, are used up in one another without residue" (4).

What Nussbaum insisted upon was the early appearance of the germ-cells *in the sexual generation of animals, i.e.* in the embryo, before this had undergone histological differentiation. In urging this he took up a very moderate attitude. To refute his conclusion from the botanical side, the conditions in the corresponding generations in the two kingdoms must be compared; that is to say, the embryo and the prothallus must be placed together, not the embryo and the sporophyte. As to Weismann's second objection, it must be insisted, that even now the early history of the germ-cells of "most animals" has as yet been very inadequately investigated. Where it has been traced back to the farthest possible point, there invariably a very early



origin has been made out. This is now so in *Moina*, *Cyclops*, *Ascaris*, *Strongylus*, *Cecidomyia*, *Chironomus*, *Sagitta*, *Phalangium*, *Lernaea*, *Micrometrus*, Scorpions (Brauer), several insects (Heymons), some sponges (Maas), Cephalopoda (V. Faussek), *Pristiurus*, *Scyllium*, and *Raja*. Until 1900 the apparent phenomena in the Vertebrata stood in the way. Here even a segmental origin of the sexual cells had been recorded in relatively late phases. This, however, though it perhaps has at present more supporters among embryologists than a morphological continuity, is only one of the ever-recurring instances of the earliest observed appearance of a thing being taken to represent its first origin. In embryological research this is only permissible when an earlier origin is absolutely out of question.

From a fair acquaintance with the embryological literature treating of the germ-cells and their origin, the writer must maintain that there is really no reliable evidence whatever pointing to the late appearance of the germ-cells in any single case. On the other hand, there is a steadily accumulating body of very strong testimony in favour of their early separation off in many different divisions of the animal kingdom. Even the case of the hydroid polypes cannot be cited in disproof, for Weismann's own great researches reveal, not so much the origin of the germ-cells in these, as their remarkable migrations.

In raising objections in face of the known facts concerning *Moina*, the dipterous insects, etc., Weismann defined, not only his own standpoint, as the upholder of an hypothetical germ-plasm, but also that of most other zoologists. Direct development implies, nay assumes, a somatic origin of germ-cells, and under its sway even an odd instance of a morphological continuity can find no place. And the exception meets with no favour until it ceases to be such and adapts itself to the rule. But, as Goethe sagely remarked (5), "die Natur geht ihren Gang, und was uns als Ausnahme erscheint ist in der Regel." And this is so simply because what we regard as the rule is often false, the real law being that with which the apparent exception conforms.

While only from two to eight (primary) germ-cells were found very early in the development of this or the other form; while, as in the higher animals, one could study the early development without seeing any germ-cells—their "segmental

origin" even being witnessed at later periods—the good old rule, in plain words, the superstition, that the offspring was formed by the union of a small portion of each of its parents, seemed to be the only logical conclusion. Thus it happened that so great an investigator and thinker as Darwin could set up his provisional hypothesis of pangenesis. When in one of the higher animals, the skate, the formation of a whole battalion of germ-cells is found to take place prior to the appearance of any trace of an embryo, a change comes over the scene: the apparent law and its exceptions exchange positions, with the consequent disappearance of the former.

Two primary germ-cells and five hundred and twelve—the latter number being that found in the development of a potentially female skate—are very different figures. In fine, in the life-cycle of the skate the origin of the germ-cells fills in so large a space as to overshadow completely everything else, and the formation of an embryo sinks into the position of being a mere incident in the life-cycle.

How little influence previous researches, treating of an early origin of germ-cells, have had upon embryological opinion, may be gathered from the following facts:—A morphological continuity of germ-cells is nowhere adopted by Weismann in his writings; indeed, until recently he accepted a somatic origin, "at any rate in certain cases"; and until the present year, 1903, which witnessed the appearance of Waldeyer's account of the germ-cells in Hertwig's "Handbuch" and the new volume of Korschelt und Heider's "Entwicklungsgeschichte," no single one of the current text-books advocated such a continuity, but one and all accepted Waldeyer's conclusions of 1870, that the germ-cells were modified pleuro-peritoneal cells (of the germinal ridges), and, therefore, somatic in origin. And it is not too much to say, that Waldeyer's conclusions (1870) as to a somatic origin of germ-cells, which he now rejects in favour of a morphological continuity, dominated all embryological opinion until the present year. This was the state of affairs when, early in 1899, the writer began his investigations. In a few cases, *Ascaris*, *Strongylus* and *Cyclops*, the track of germinal continuity had been followed by Boveri, Haecker, Rueckert and Spemann for the first five cleavages of the egg. They had not got beyond this point, for under the erroneous conception of direct development

they had assumed the first products of the cleavage to be embryonic in destiny, and in the cases of *Ascaris* and *Strongylus* the subsequent development after the formation of the primitive germ-cell was, and still is, unknown. How little claim these researches, important though they be, can have to have demonstrated a morphological continuity of germ-cells is shown by the circumstance that it cannot be stated with any degree of certainty whether the organisms dealt with were merely larval (asexual generations) or true "embryos" of a sexual generation. Our knowledge of the development of Nematodes, which we mainly owe to Maupas, and the analogy of other cases, *Nereis*, etc., point to the former as the real truth.

## II. THE HISTORY OF THE GERM-CELLS IN ELASMOBRANCH FISHES.

The material so far employed in the researches has consisted of the smooth skate (*Raja batis*), the two dogfishes (*Scyllium canicula* and *Pristiurus melanostomus*), some embryos of the spiny dogfish (*Acanthias*), chicks, etc. The chick, which was the animal employed by Waldeyer and others, is not a favourable object for research upon the germ-cells. It can, however, easily be made out in it that they are not somatic in origin, and that they migrate to their ultimate positions in the germinal ridges or nidus. As yet no attempt has been made to follow out the history of the germ-cells of any mammal, for the technical difficulties in the way of research here are very great. But it cannot for a moment be doubted that, *mutatis mutandis*, what has been found to hold good for the lower vertebrates will also apply to the higher ones.

On taking an early Elasmobranch embryo, and staining this with certain reagents (osmic acid or Heidenhain's iron-hæmatoxylin), the germ-cells within it are rendered so prominent that one can even count them. These germ-cells are seen to be cells of large size, about 0.02 mm. (Figs. 1 and 2), whereas even the largest somatic cells do not exceed 0.012 to 0.014 mm. Their cytoplasm has a peculiar glassy character, and possesses little or no affinity for any of the ordinary dyes. The nucleus appears to be invariably of the twin or bilobed character, described by Rueckert and Haecker in *Cyclops* some years ago, and now known

to be present in the germ-cells of very many animals—in all those, in fact, in which it has been sought after. Within the cytoplasm for long periods of the development there are yolk-plates, of a shape more or less characteristic of each species. These plates are most numerous in the early periods, their presence is one of the signs pointing to an origin of the cells from the cleavage, and they are gradually used up as development proceeds, until by the time the critical period<sup>1</sup> is approaching they are all gone. These plates stain a jet-black with iron-hæmatoxylin, and this betrays a germ-cell at once wherever it may be. But of course, in identifying such a cell as germinal, one must take other characters into account. So long as the germ-cells of any embryo possess yolk plates, they remain in a state of rest and undergo no increase in number. In *Scyllium* this lasts until the embryo is 25-26 mm. in length, the critical period being here reached with 32-33 mm., in *Raja* until upwards of 50 mm., the critical period being attained at 64-66 mm. The result is that the original germ-cells, termed by me the *primary germ-cells*, excepting any which may degenerate, remain constant in number<sup>2</sup> for a very long period of the development. Their products, when they do divide, are termed *secondary germ-cells*. With the first formation of these in the meantime my researches close, but the subsequent history up to the ripening of the

<sup>1</sup> In two memoirs, "Certain Problems of Vertebrate Embryology" and "The Span of Gestation," the nature and peculiarities of the critical period have been dealt with in full. This period is that at which "the embryo" is first present in all its parts, and when it is first beginning to resemble the form whose offspring it is. It is at this epoch that the sex is first announced, that the embryo first begins to nourish itself (by digesting yolk or by forming an allantoic placenta, etc.), and that larval structures begin to degenerate. Though even now the existence of this epoch is still ignored by embryologists in general, this circumstance in no way lessens its importance. It is only in embryological text-books and in the researches of upholders of epigenesis and direct development that the peculiarities of the critical period meet with no appreciation. But Nature herself is unable to accomplish that which embryologists find so easy, the abolition of the critical period!

<sup>2</sup> In a recent work upon "Practical Embryology," Minot characteristically states that only the germ-cells of *Acanthias* have as yet been investigated! Like nearly every fact concerning the germ-cells of Elasmobranchs described in the present writing, the constancy in number escaped the notice of Woods, the pupil to whose work Minot refers. Indeed, beyond the demonstration of the glaring inaccuracy of Minot's dogmatic assertions concerning the germ-cells, which were never based in observation, it is hard to say what Woods' work really proves. Woods' paper appeared on May 26th, 1902, while the writer's first account of the germ-cells of *Raja* was published at the close of 1900.

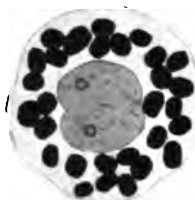


FIG. 1.



FIG. 2.

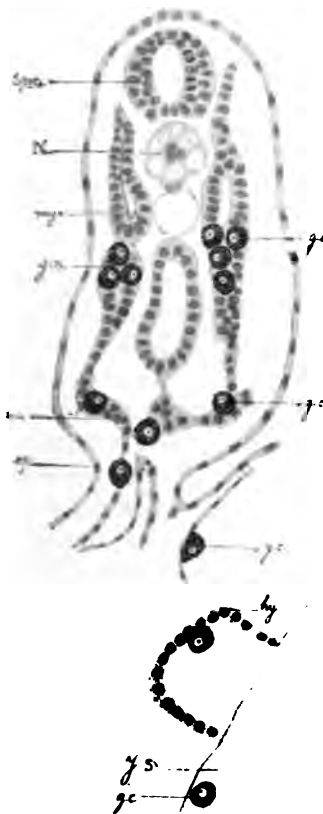


FIG. 3.

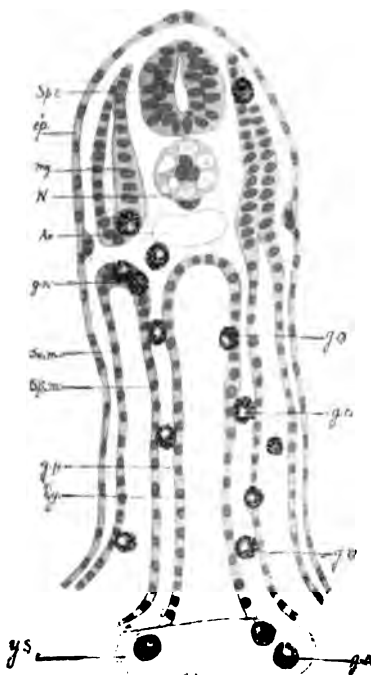


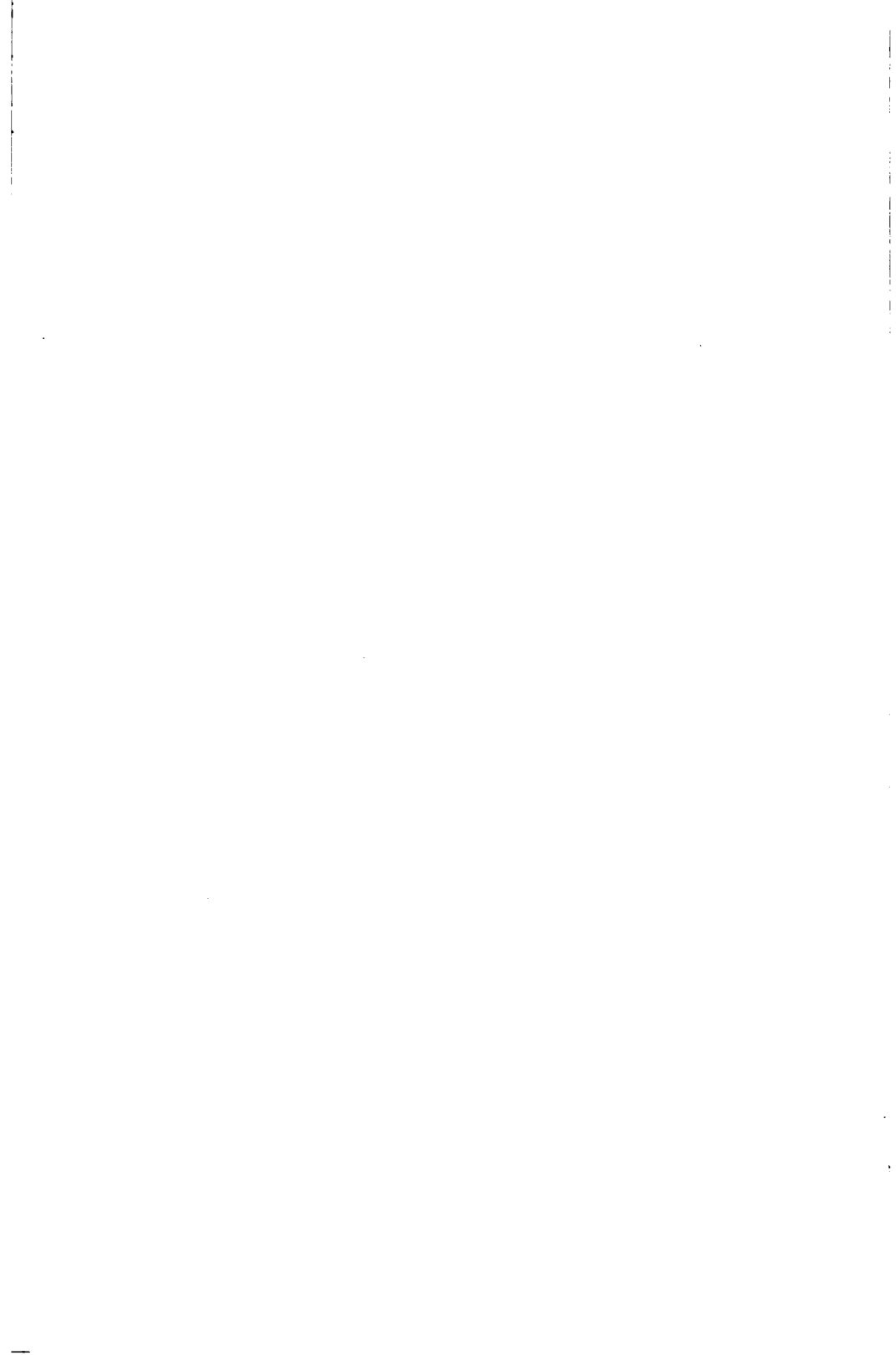
FIG. 4.

FIG. 1.—Primary germ-cell of *Pristiurus melanostomus*. The cytoplasm is glassy in character and contains a large number of (blackened) yolk-plates. The nucleus exhibits duplication, i.e. autonomy of paternal and maternal portions.

FIG. 2.—Primary germ-cell of the skate, *Raja batia*.

FIG. 3.—The conditions seen in two transverse sections (the 39th and 41st sections of a row) of a 4½ mm. embryo of *Pristiurus*. The lettering is as follows: sp. c.=spinal cord, n.=notochord, my.=myotome, g. n.=germinal nidus, me.=mesoblast, ep.=epiblast, hy.=hypoblast, y. s.=yolk-sac, g. c.=germ-cell.

FIG. 4.—A diagrammatic section of an early skate-embryo. To illustrate the migrations of the germ-cells along the germinal path, g. p., and showing germ-cells in various abnormal situations. The lettering is as in Fig. 3, excepting so. m.=somatic mesoblast, sp. m.=splanchnic mesoblast, ao.=aorta.



sexual products has been followed out very fully by Balfour and Semper, while we owe further details as to oogenesis and spermatogenesis in Elasmobranchs to the work of Rueckert and Moore.

The observations originally began with the tedious but instructive counting and tabulation of the primary germ-cells of skate embryos of about 30 mm. This work is anything but pleasant, albeit it has occupied much time during the last four years, and the writer can perhaps flatter himself that he has now counted and tabulated the germ-cells of more embryos of various species than any other investigator is likely to do for some time to come. From the counting and tabulation, two results among others stand out prominently. These are, that the number of primary germ-cells is a constant quantity for the species, and that during long periods of the development, in *Pristiurus* in embryos from 4 mm. to 20 mm., in *Raja* from 6 mm. to 42 mm., no single embryo ever has all its germ-cells in the normal position.

As to the bearings of these results, the first is undoubtedly extremely important in aiding in the conception of a morphological continuity, while the second, though it has no direct bearings upon this question, helps to undermine and destroy the view of a somatic origin of germ-cells. If there be a somatic origin of germ-cells it must be possible for them to arise anywhere and everywhere in the body, for there is hardly a single organ in the whole body free from the risk of possible "infection" by such aberrant or vagrant germ-cells.

The counting of the germ-cells can only be carried out with any degree of accuracy in what, under modern microscopical methods of research, are fairly thick transverse sections. I incline to think that the resulting total is always, or nearly always, somewhat too high. It is rarely, if ever, too low; for it is far easier to make the mistake of counting a germ-cell twice, than it is to overlook one. At the best, even when one has obtained a theoretical number, there may have been one or two more, which have degenerated, or failed to enter the embryo, or these may still be in the yolk-stalk or yolk. In many instances, especially in *Pristiurus* embryos, from two to eight germ-cells have been encountered in the yolk-stalk, and in very early

embryos some of them may lie either in the upper part of the yolk, or anywhere upwards from it to the level of the germinal nidus and higher. In text-figure 3 the appearances seen in two neighbouring sections of an embryo of *Pristiurus* (No. 47 = 4½ mm.) are depicted, and in it is shown vividly the "line of march" of the germ-cells from the yolk-sac, y.s., to the germinal nidus, g.n.

For questions relating to the enumeration, therefore, it is necessary to deal only with embryos in which the migrations of the germ-cells are finished. In the skate such embryos are those from 16 to 42 mm., in *Pristiurus* those from 5 mm. to 20 mm. or so. The very first embryo in which the germ-cells were closely studied, *R. batis*, No. 454, showed them so brilliantly marked off, like black beads of large size, from all the other cells, that the idea suggested itself of attempting to count them. The total came to 512. I now know that this number cannot have been absolutely correct; but, as in other and later cases, the "error of observation" is probably not usually greater than about equal to the number of germ-cells not really in the embryo but in the yolk or in degeneration. Some other instances also gave approximations to 512, while still others yielded a different total, about 256. As big eggs and little eggs, respectively female and male in destination, were already known in *Hydatina senta*, *Dinophilus gyrocolliatus*, and *Phylloxera coccinia*, a natural suspicion arose that something of the same sort was at the bottom of the two numbers, and this was confirmed by the circumstance that every case taken seemed to approximate to 256 or 512. How much labour the solution of a trifling problem of embryological research may entail, may be gathered from the statement that it took three months of hard work to establish beyond question the fact, that potential male embryos of *R. batis* possess a number of germ-cells approximating to 256, potential females to 512. Without at this juncture entering further into the matter, it may be added, that this find was the starting-point of a further investigation into the determination of sex.

It was then found that in *Scyllium* and *Pristiurus* the number of primary germ-cells approximated to 128. Thus, for 30 embryos of the latter the average total of 126.73 was obtained. As the theoretical original number was 128, and as



one of these had gone to the unfolding of an embryo (to contain the rest), the actual find here is practically identical with the theoretical number. From these and other cases it was concluded that the number of the primary germ-cells was always one of the series, 2, 4, 8, 16, 32, 64, 128, 256, 512, etc. *minus* 1, or  $2^n - 1$ . In passing, it may be added that in certain abnormal embryos either very few or practically no normally-placed germ-cells were found either upon one or upon both sides of the body, and once more this points to their origin outside the embryonic body. This is certain; that is to say, it can be established, that the germ-cells arise before there is any embryo, and that on its formation they migrate into it along the connection between embryo and yolk-sac, the yolk-stalk. These migrations begin very early, even before the closure of the medullary plate, and they do not go on for any great length of time. In *Pristiurus* the germ-cells are most easily found in migration in embryonic foundations of 2 - 4.5 mm., in *Raja batis* in such of 4 - 8 mm.

Owing to these migrations from the yolk-sac and blastoderm, it never happens that all the germ-cells find their way into the normal position, the germinal nidus or its future site, but a percentage of them (25-30% in the skate, 10-12% in *Pristiurus*) comes to lie in one or other of a great number of places really foreign to them. Thus, sometimes they may be found in the head, head-somites, brain, the gill-region, the skin of the trunk or head, the pericardium, the liver, even occasionally in the blood, the kidney-tubules, the body-cavity, myotomes, spinal cord, gut-epithelium, especially of the yolk-stalk and of the rectum, etc., etc.—in fine, there is hardly a place in the whole trunk or head in which such aberrant germ-cells have not been observed. (Compare the diagrammatic figure, Fig. 4.) For a long time the notochord was one such exception, but finally one of my pupils found a germ-cell even there. At the moment of writing the thymus and thyroid are about the only places in which vagrant germ-cells have not been encountered. Usually they remain in such places, often becoming encapsulated; at all events these things are true of those in the older embryos, and many of them, whether or not the majority cannot be stated, degenerate there. The phenomena of their degeneration by simple atrophy have been described elsewhere. Regarded in the

light of other results upon the life-cycle and upon the mode of development of "the embryo" from one such primary germ-cell, there is a strong temptation, which the writer has not resisted, to bring such aberrant germ-cells into genetic relationships with the tumours and carcinoma. (Compare the list of literature.)

But the primary germ-cells can be carried back to still earlier periods of the development. In the later phases of the cleavage their identification is not a matter of great difficulty; and from the study of the cleavage, the size and constitution of the blastoderm at fertilisation, and from the sizes of the primary germ-cells themselves, it can be shown that the primary germ-cells date back to a cell of about the tenth cleavage phase as the primitive germ-cell (U.K.Z. of the diagram). In the skate, *Scyllium* and *Pristiurus*, there are about ten divisions of the egg before the appearance of U.K.Z., the primitive germ-cell, and of this latter there are seven further mitoses in *Scyllium* and *Pristiurus*, eight in potentially male skate, and nine in potential females.

These data are so important that it may be of interest to give some details respecting them. We have already seen that the primary germ-cells have an average diameter of 0.02 mm., and that their number is a constant one. Assuming them to have arisen in the course of the cleavage, it may be asked, "from a cell of what size can they have taken their birth?" The microscopical sections of germ-cells are, of course, segments of spheres of a radius of 0.01 mm. From these data it is not difficult to calculate out the size of a sphere which by regular equal divisions would ultimately give as its products, for instance, 128 spheres of 0.01 mm. radius. Moreover, the germinal disc of an Elasmobranch egg, and this is the real functional egg which alone undergoes cleavage, is practically a hemisphere of a certain radius. If, starting again from the size of the primitive germ-cell, further calculations be made, we soon reach the size of hemisphere corresponding to the original germinal disc, whose size we know. In this way tables of the following kind are obtained :—

Ascending series of spheres of double volume, calculated from the starting-point of 0.01 mm. radius by Dr J. H. Ashworth.<sup>1</sup>

	Radius mm.	Diameter mm.	Skate. ♀	Germ-cells. Skate. ♂	<i>Scyllium</i> .
1.	0.01	0.02	512	256	128
2.	0.0125	0.025	256	128	64
3.	0.0159	0.031	128	64	32
4.	0.020165	0.04	64	32	16
5.	0.0252	0.05	32	16	8
6.	0.0314	0.062	16	8	4
7.	0.039	0.078	8	4	2
8.	0.049	0.098	4	2	1 = U.K.Z.
9.	0.058	0.116	2	1 = U.K.Z.	(1)
10.	0.061	0.1234	1 = U.K.Z.	(1)	(2)
11.	0.076	0.1536	(1)	(2)	(3)
12.	0.096	0.1934	(2)	(3)	(4)
13.	0.12058	0.24	(3)	(4)	(5)
14.	0.1512	0.30	(4)	(5)	(6)
15.	0.189	0.378	(5)	(6)	(7)
16.	0.2394	0.478	(6)	(7)	(8)
17.	0.302	0.6	(7)	(8)	(9)
18.	0.378	0.75	(8)	(9)	(10)
19.	0.478	0.95	(9)	(10)	
20.	0.6	1.2	(10)		
21.	0.75	1.5			

The interval between each line in the table represents a mitosis. The first column gives the key to the number of mitoses in any form. The second and third columns show the sizes of the radii and diameters respectively of the cleavage segments (spheres) formed. The remaining three columns give the cleavage in the female skate, the male skate, and in *Scyllium*. Thus, in the female skate there are 512 primary germ-cells, and these are seen to go back to a cell of 0.1234 mm. diameter as the primitive germ-cell. The blastoderm has a diameter of 1.5 mm., but this is a hemisphere, and, therefore, in the table the

<sup>1</sup> This table is given as a preliminary result. Dr Ashworth and the writer intend to make a further examination and control of the figures and measurements.

cleavage of the female skate seems to start at line 20 with a sphere of 1.2 mm. From U.K.Z. to this point there are 10 divisions, and, therefore, in the development of the female skate there are to the formation of the primary germ-cells from the commencement of the cleavage in all 19 mitoses. In the development of the male skate there are only 256 primary germ-cells of 0.02 mm., and therefore one cleavage less; but here the disc at the beginning of cleavage is less than in the female skate, having in fact a diameter of 1.2 mm. (hemisphere). On the other hand, in *Scyllium* there are 128 germ-cells; these start from a primitive germ-cell of 0.098 mm. diameter, and the original blastoderm has a diameter of 0.95 mm. (hemisphere). In *Torpedo ocellata* the germ-cells are smaller than in *Raja* and *Pristiurus*, and they are comparable in size to the products of the first division of the germ-cells of the latter. Apparently they have gone through an additional division, but, although their number is not yet known, from their sizes and from that of the blastoderm, which is smaller than that of *Pristiurus*, it is not difficult to unravel the facts. The blastoderm of *Torpedo* has a diameter of 0.75 mm., the primitive germ-cell probably has a diameter of 0.078 mm., and, as likely, there are 128 primary germ-cells. In this form the germ-cells have not yet been counted; because, although they possess yolk while outside the embryonic body, they would seem to lose this soon after they enter the embryo. In this way they become lost to sight.

The facts just described tempt one to make a slight digression. The number of cell-divisions, from any given point in the life-cycle to any other given point, in all probability is for any given species a constant quantity. This is a matter of some importance, as will appear later. In man it is not known, nor soon likely to be, how many divisions of the egg (zygote) take place before the appearance of U.K.Z., the primitive germ-cell. Allan Thomson calculated that in one ovary of a new-born infant there were about 70,000 eggs (oocytes). As one might expect, this number is somewhat too high, the real maximum being about 65,000 at most. There are here, therefore, not more than 16 divisions from the primitive germ-cell to the formation of the oocytes, and it is, perhaps, not a matter of very great import at the moment, in which of these the primary germ-cells appear. As to the primitive germ-cell here, I incline to the

belief that it is formed not sooner than the third or fourth cleavage and not later than the tenth.

### III. THE METAZOAN LIFE-CYCLE

The things briefly described in the preceding chapter furnish the key to the problem of the Metazoan life-cycle and mode of development. For facts such as these the writer had, without having any idea of their real nature, searched for years again and again, but in vain. As long as it was possible to maintain the doctrine of a somatic origin of germ-cells, there appeared some reason for regarding the mode of the development as direct; and, though certain phenomena pointed significantly enough to an antithetic alternation of generations, under the view of a somatic origin of germ-cells it could not be demonstrated that an antithetic alternation must of necessity underlie the facts.

With the finds relating to the primary germ-cells it at length became possible to fill in a portion of the diagram (Fig. 5), which in former years had been a complete enigma. It was long clear that "the embryo" at a certain epoch, the critical period, began to suppress the asexual foundation, the phorozoön, upon which it had arisen. But how, and out of what, an embryo had been evolved, was the greatest puzzle which in embryological research I have ever encountered. It had, indeed, long been foreseen that something resembling the formation of spore-mother-cells in the Metaphyta was demanded, if a cycle comparable in any way to that obtaining in the vegetable kingdom underlay the development. As already stated, this was on investigation missing, because it was not searched for in the right place.

But now it is at length quite clear that the formation of the primary germ-cells (Fig. 5) in the skate—and in all probability in every other Metazoön, for it is not possible that two fundamentally different modes of Metazoan development should exist—corresponds broadly to the genesis of spore-mother-cells on the asexual generation of a plant, the sporophyte. With this recognition the basis of a comparison between the phenomena of the life-cycles of the Metazoa and the Metaphyta, so as to show their essential similarity, is afforded. In the same way, the discovery of the formation of the primary germ-cells, and of the

epoch of their coming into being, throw new and unexpected light on the course and nature of heredity, as well as upon variation.

These are the chief results of my work on the germ-cells; and, though other and doubtless important finds have been made, the latter sink into insignificance when placed beside the former.

Certain parts of the diagrammatic figure (Fig. 5) have been adopted, as already stated, from the writings of other embryologists. This, however, has not been done without important modifications, for which the writer is alone responsible. Originally, towards the close of 1900, Boveri's diagrams of oogenesis and spermatogenesis formed and filled in portions of the life-cycle. Doubts arose as to their completeness, in view of the existence of two sorts of eggs and two kinds of sperms in certain cases, and the working-out of the probable course of oogenesis in the skate finally resulted in the modifications here depicted. The first part of the figure, from the zygote, Z., formed by the union of egg and sperm, to the primitive germ-cell, U.K.Z. (the "Ur-Keimzelle" of German authors), is from Boveri's and Weismann's figures. Their diagrams and conclusions, however, go no further than U.K.Z., and in their figures the line from Z. to U.K.Z. marks what Weismann terms "the germinal track" (Keimbahn), and the products to the left of it are assumed to be cells of the embryo! As in the skate or other Elasmobranch there is no possibility of the existence of any part of an embryo prior to the formation of U.K.Z. it is out of question that the said cells can be part of this. It is a pure assumption that they are parts of an embryo; and in *Ascaris megaloccephala*, for instance, to which Boveri's identical diagram refers, it has never been established—and be it added, it never will be determined—that directly from the cleavage of the fertilised egg the sexual generation or embryo, as it occurs in the horse, takes its origin. The later history is here unknown. If it be imagined possible that here, by direct development, the sexual form of *Ascaris*, as it is found in the horse, can arise, a reference to the account of Maupas' results (6) of investigations into the life-histories of a number of Nematoda will dissolve the illusion. Indeed, it may safely be predicted that, when the facts become known, of the two primary germ-cells of *Ascaris*, formed by division of the cell U.K.Z., the one will be seen to form an embryo or sexual generation, while the



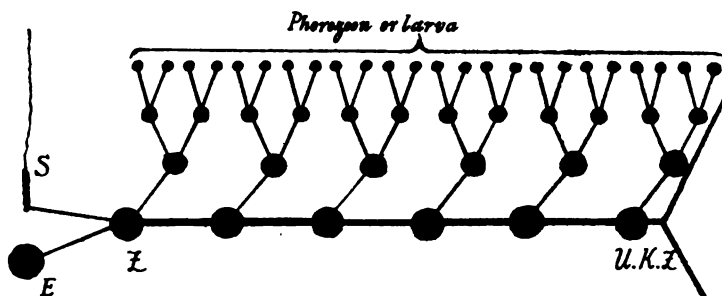
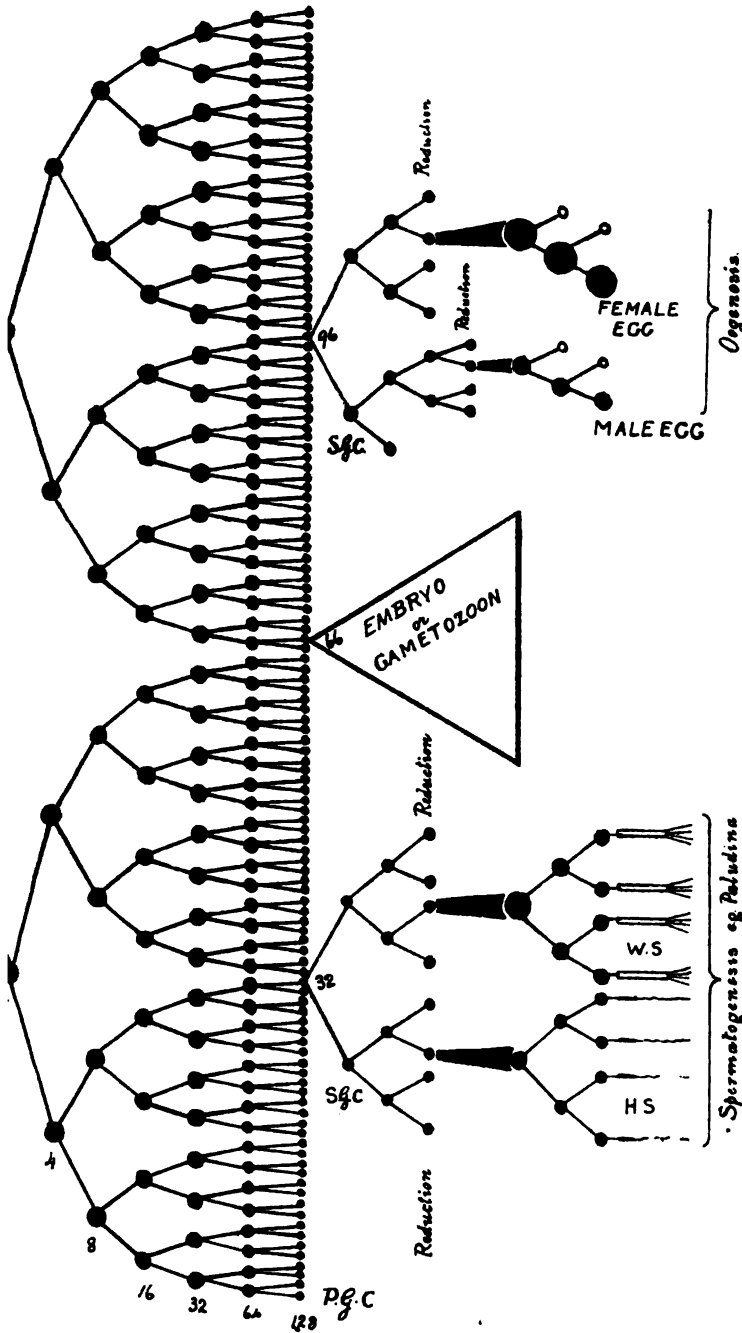


FIG. 5.—Diagram of the life-cycle of the skate, *Raja batris*, or of *Scyllium* or *Pristiurus*, illustrating the union of the egg and sperm, E. and S., to form the zygote, Z., the origin of the phorozoan or asexual generation, the germinal track from Z. to U.K.Z., the latter being the primitive germ-cell. The divisions of the latter are carried to seven mitoses, as in *Scyllium* or *Pristiurus*, giving 128 primary germ-cells at P.G.C., instead of the full number of nine divisions in a potentially female skate, yielding 512 primary germ-cells. Diagrammatically the unfolding of one primary germ-cell, the 66th, is depicted as forming an embryo or gametozoan. To complete the track of heredity from generation to generation through the morphological continuity of the germ-cells a diagram of oogenesis has been appended to the 96th germ-cell, one of spermatogenesis to the 32nd. In the former, the formation of a male-egg and of a female-egg is shown, in the latter the two forms of sperms (in *Paludina* after the statements of Meves), i.e. the hairlike or functional, and the wormlike or functionless spermatozoa.







other will furnish its sexual products. It will, doubtless, be urged that on my part also it is an assumption that the cells to the left of the line Z. to U.K.Z. give origin to the larva or phorozoon. In a sense this is true; but the one supposition is *prima facie* as good as the other, and on the further evidences to be adduced it is a good deal better. Moreover, *it has never yet been demonstrated for any animal whatsoever that the first cleavage products go towards the formation of an embryo, whilst it has often enough been proved up to the hilt that in many instances in various divisions of the animal kingdom they are concerned in forming parts of a series of temporary and transient organs, making up the larva, not in the least identical with or comparable to an embryo.*<sup>1</sup>

From the existence of a transient nervous apparatus, a blastoderm, and other evanescent structures, the conclusion was long ago arrived at that there was a phorozoon or asexual generation in the life-cycle of the skate, etc. From all the known facts of embryology such a phorozoon cannot arise out of an embryo; *it must precede an embryo*. There is no embryo by the time the period P.G.C. of the figure is reached, the formation of such commencing here. Therefore, the first products of the cleavage—up to the tenth division—apart from the line leading to U.K.Z. must be the phorozoon, or asexual generation. There is abundant testimony of the truth of this conclusion in the literature of embryology, but evidence from another side will be found in, for instance, E. B. Wilson's published researches upon the cell-lineage and development of *Nereis*.

There was at first some hesitation in the writer's mind as to the possibility of using Wilson's results (7) in support of the view here presented of the nature and destiny of the first cleavage products. A perusal of the lecture cited below served to remove this. His work of 1892 and his more recent results must be taken together, for Wilson himself has seen reason to alter his earlier interpretation in some slight but important

<sup>1</sup> In the best investigated cases, that is to say, in ALL the instances which have been closely studied, it is established that the products to the left of the line are all larval or asexual in destination, and that they do not go to form parts of the embryo. Moreover, until the publication (Jan. 1902) of my figure, nothing whatever was known of the subsequent history after U.K.Z. down to the formation of the primary germ-cells. That is to say, Boveri's and Weismann's figures, if taken as showing either the course of the life-cycle or the track of heredity, are like the play of "Hamlet" with the chief character omitted!



products form the two "mesoderm-bands," *i.e.* the foundations of the worm. In contra-distinction to other authors, the writer must maintain the opinion that M. is a primary germ-cell, and that it gives rise to the whole of the sexual generation, in this case the worm. In this connection it may be of interest to recall the circumstance that long ago Hatschek expressed the view that the two products of M., the well-known "pole-cells" of Hatschek, were originally eggs. This suggestion has been criticised by Kleinenberg. If the pole-mesoderm-cells be not "eggs"—and most certainly they are not—they at least arise by the division of the next thing to an egg, a primary germ-cell. Wilson, on the other hand, approves of E. Meyer's amendment of Hatschek's view into a correspondence of the mesodermal bands with—paired gonads! This is multiplication of causes with a vengeance! In the restricted sense indicated above there is something to be said in favour of Hatschek's interpretation: the other idea is wildly impossible.

In *Nereis* the further destination of the remaining primary germ-cell D is unknown, beyond that it comes to form part of (*i.e.* to lie in) the hypoblast. There is no difficulty about this. Even in the skate many of the primary germ-cells may for a time lie in the hypoblast, but they do not give rise to hypoblastic cells. As Wilson remarks, "the ultimate court of appeal . . . lies in the fate of the cells" (*loc. cit.*, ii. p. 41).

Another apparent difficulty, more especially to the view of the complete similarity and equivalence of the primary germ-cells, would be that possibly sometimes the embryonic cell, the somatoblast, may exceed the primary germ-cells in size. But this very difference in size—assuming it exist—may serve to explain why some particular primary germ-cell is chosen to unfold as an embryo instead of some other. Position alone cannot always be at the bottom of this. In the skate, for example, the embryo does not invariably begin to arise at one certain spot upon the blastoderm. It may be that the stimulus afforded by an extra amount of food-yolk may have much to do with the initiation of development.

Very suggestive and significant, in the light of my researches upon the germ-cells, are the following passages from E. B. Wilson's *Nereis* memoir. Statements equally pregnant with

meaning will be found in Eisig's work on the development of *Capitella* (8). On p. 393 Wilson writes: "Transition to the Bilateral Period.—As far as the development of the permanent organs is concerned, the transition from the spiral to the bilateral type of development is remarkably abrupt." It may be mentioned that at the close of the spiral period there are, according to Wilson, 38 blastomeres present. That is to say, the majority of them are products of the fifth cleavage.

On p. 444 he asks: "What is the significance of the spiral and bilateral forms of cleavage, and where lie the causes that determine the transformation of the one into the other?" Further on he writes: "The most striking feature in the cleavage, and the one on which the entire discussion may be made to turn, is the sudden appearance of bilateral symmetry in the cleavage. The meaning of the bilateral cleavages in themselves is perfectly obvious. They are the forerunners of the bilateral arrangement of parts in the adult; and, as such, their explanation belongs to the general problem of bilateral symmetry, which need not be considered here. The all-important point is that the bilaterality does not appear at the beginning of development. It appears only at a comparatively late stage, and by a change so abrupt and striking as to possess an absolutely dramatic interest." And so on. I refrain from further quotation because Wilson's work contains no real solution of the problem. To my mind the solution was lacking because, on the one hand, it was not recognised that the mode of development was by means of an alternation of generations; and, on the other, the history of the primary germ-cells of *Nereis* was, and still is, unknown.

If the reader will compare Wilson's statements with the course of development depicted in my diagram—not forgetting, I trust, that the latter is a diagram and nothing more—the meaning of the spiral cleavage and of the sudden and abrupt change, of which Wilson writes, may become apparent. The apical mode of growth, so characteristic of the early formation of the asexual generation in both plants and animals, and which is retained for the whole life-span of the sporophyte of plants, might also be described as spiral. Indeed, it is so regarded and described by botanists. With the cutting-off of the connection between the primitive germ-cell and the asexual generation or

phorozoön, we witness the practical end<sup>1</sup> of the spiral mode of cleavage and the commencement of the bilateral period. With this the formation of the primary germ-cells is connected; and, following the genesis of these, a start is made in the unfolding of an embryo. In this way my diagram gives a general interpretation of Wilson's finds, not to mention those of other observers. And thus the phenomena, observed in the development of *Nereis*, are seen to be due to an antithetic alternation of generations, where the asexual generation arises in a spiral or apical manner, where the sexual generation is characterised by a bilateral mode of formation, and, lastly, where the formation of a primitive germ-cell, and of "x" primary germ-cells from this, may be predicted between the two generations; that is to say, prior to the development of the sexual generation.<sup>2</sup>

In the course of all the years spent in the attempt to unravel the mode of Metazoan development, at various times many things have seemed inexplicable; but, wherever their history has been discovered—and with patience and pertinacity this has usually happened—they have been found to fit into an antithetic alternation of generations, and into nothing else. If Wilson's finds be not based in such an alternation, but be in connection with a "direct" mode of development, they would seem to include facts which will never be explicable, for logically such a round-about kind of development cannot be termed "direct." Or, shall we explain and describe them as the development of the Scyphozoa is recited in almost all the current text-books, by the omission of any reference to the main portion of the asexual generation, the creeping stolon, discovered by Sars (9)? Such a course may simplify matters, but it hardly makes for the discovery of the facts of nature!

Reverting to the diagram of the life-cycle of the skate, at present it is possible only by comparison and induction to show the fate of the cells to the left of the "germinal track" as far as U.K.Z., the primitive germ-cell. The comparison with other

<sup>1</sup> The practical end, but not the actual termination; for, as Wilson points out, "it is only in the peculiar changes involved in the formation of a larval organ, the prototroch, that the spiral form of division overlaps the bilateral period" (*op. cit.*, p. 393). The "prototroch" is a circle of cells from which the preoral ciliated band arises.

<sup>2</sup> It is, perhaps, possible that in some animals, e.g. Annelida, the conditions at P.G.C. in Fig. 5 would be more accurately represented by a rotation of the line of P.G.C. through a right angle.

cases only goes to prove its correctness, and, I am convinced, the number of such will increase in the proportion as the study of cell-lineage, so ably established by Whitman, Mark, and E. B. Wilson, replaces the bootless pursuit of the three sacred layers of embryologists.

Up to the point U.K.Z. of the diagram the germinal track in Weismann's sense lies apparently in the larva. It may be objected that in making this substitution in favour of the phorozoon, the embryo has been displaced, and that the germinal track is here somatic. The reply is that the cell U.K.Z. and its immediate ancestors never form part of the larva, and the period from Z. to U.K.Z.—no matter how long it be, whether four cell-generations as in many Invertebrata, or ten as in the skate, etc., or four thousand—is marked by a mode of growth and cell-division conspicuous by absence in other parts of the diagram.<sup>1</sup>

This statement requires both elucidation and emphasis. The mode of growth of the sporophyte in plants is essentially apical; that is to say, wherever there is an apex there are always one or more apical cells, which by their division give off products towards the centre. In the sexual generation of a Metazoon the mode of growth differs *in toto* from this; for here all the products ultimately undergo differentiation, and embryonic or germ-material, corresponding to apical cells, has no existence. The older embryologists of the first half of the nineteenth century thought differently, and some pathologists still cling to their views, but these have no shadow of foundation in fact. And whenever a *neoplasm* or tumour may appear to have arisen by the formation of one or many germ-cells from somatic cells in any part of the body, as quite recently maintained for sarcoma and carcinoma, the real source of its origin will have been an encapsulated vagrant primary germ-cell.

The initial mode of growth and formation of the asexual generation or phorozoon in animals—an organism never of a very high degree of organisation—is entirely comparable to that of a

<sup>1</sup> Spemann has already compared the mode of origin of the first cleavage products in Nematodes, more especially in *Strongylus*, to the apical mode of growth in the sporophyte of a plant. He notes that the cell along the line Z.-U.K.Z. in my diagram acts as though it were an apical cell of a sporophyte ("Die Entwicklung von *Strongylus paradoxus*," in *Zool. Jahrb. Morph. Abteil.*, vol. viii, p. 304, 1894-95). This author, one of the ablest of younger embryologists, almost, indeed, anticipated the discovery of a morphological continuity—to a much greater degree than Boveri and Weismann, to whom Waldeyer gives the credit.



sporophyte. As in simple cases of the latter, there is here one "apical cell," which never itself forms part of the phorozoon, but instead thereof gives off into the latter a greater or less number of products, while retaining its own unicellular or Protozoan character. Nor would the conditions be altered, were there several growing points, as generally met with among the Hydrozoa.<sup>1</sup>

Sooner or later upon the phorozoon the primitive germ-cell enters into activity. It may divide before the phorozoon is properly differentiated, as nowadays is certainly the case in many instances, or, theoretically, its divisions may happen at a later period. These divisions, however, must, of necessity, precede the formation of an embryo or sexual generation. In the skate and *Pristiurus*, etc., the divisions of the primitive germ-cell which give birth to the primary germ-cells, take place before the phorozoon is fully differentiated, and, of course, before there is any trace of an embryo. In all those Elasmobranch fishes yet studied by me, the commencement of the divisions of the primitive germ-cell would appear to date from about the tenth cleavage phase, and in the skate there are either eight (male) or nine (female) divisions.

In the portion of the diagram showing the origin of the primary germ-cells, these have only been drawn to seven divisions, giving one hundred and twenty-eight. To exactly embrace the full significance of the discovery, the drawing ought to include two further divisions, yielding five hundred and twelve germ-cells at P.G.C. That is to say, to represent accurately the conditions in *Raja batis*, No. 454, for example, the diagram ought to be at P.G.C. four times as wide as it is at present.<sup>2</sup> Its width is about 16½ centimetres; it ought to be about 66.

<sup>1</sup> It should be mentioned that de Vries and Weismann have already noted the resemblance in mode of growth between the sporophyte and the colonial Hydrozoa. Many of the latter also possess the indefinite unrestricted power of growth, so characteristic of the sporophyte of the higher plants. As a rule the asexual generations of the higher Metazoa do not exhibit this faculty. They rarely obtain a chance of showing it, for it is their usual fate to undergo early suppression by the sexual generation. When, as happens sometimes in the human subject, either no embryo is formed, or it degenerates or aborts prior to or at the critical period—a common epoch of abortions—the asexual generation, the trophoblast or chorion, may go on growing indefinitely, if left in the uterus (hydatid mole, chorio-epithelioma).

<sup>2</sup> This fact must be borne in mind, in order to appreciate fully the true bearings of the finds upon problems of heredity and variation. At the time a female skate embryo, for example, commences to develop, there are about 500 understudies of it in existence.

The division of the primitive germ-cell into primary germ-cells is a well-marked epoch in the life-cycle, and one of the greatest possible moment. Hitherto its import has been overlooked by every embryologist, and its record was made for the first time as one of the results of the *Raja*-researches. From every point of view it is as important as the phenomena to which embryologists attach the term "maturation," and probably its essential necessity in development will not need to wait long for ample recognition in Embryology.

The number of the products of the primitive germ-cell is very large in the skate—as many as 512. But it must be pointed out, that this number furnishes no criterion for other animals. There may be cases in which it is larger, though, I imagine, the occurrence of many such is unlikely. Undoubtedly there are instances in which it is much smaller; and probably these are well represented among the Invertebrata. In short, it may be as low as two; but, as the sexual generation or embryo must arise from one product, and as this must contain some sexual elements, it can never be lower than two. In other words, the primitive germ-cell must divide at least once, yielding two primary germ-cells, of which one will give rise to an embryo, and the other will supply the "sexual products." Apparently, it divides once in *Cyclops* and *Ascaris megalocephala*, twice in *Cecidomyia*, and thrice in *Chironomus*.

As to the rest of the diagram, this relates to the divisions of the primary germ-cells into secondary germ-cells within the embryo, the determination of sex, and the final phases of oogenesis and spermatogenesis. As already stated, the primary germ-cells remain quiescent during the unfolding of the parts of the embryo, and just prior to the critical period they commence to divide and form secondary germ-cells. Of the number of these divisions in any form, nothing is known, but it is certainly always a definite one, though not necessarily the like one in both sexes. Indeed, it is almost certain, that a germ-cell, whose ultimate products will become male eggs, divides at least once oftener than one whose destiny it is to furnish female eggs. It is also of importance to note that the divisions of the primary germ-cells precede the revelation of the sex of the embryo, and in some way or other are connected with it. The final products of the secondary germ-cells are termed oogonia and sper-

matogonia respectively. In the final division of these into oocytes and spermatocytes as described in a later chapter, the important phenomenon of the reduction of chromosomes takes place. As will be seen in other chapters, this phenomenon underlies the determination of sex, and all the phenomena of heredity, etc.

With the exception of the portions of the diagram relating to the determination of sex, the data concerning oogenesis, etc., are taken, as will be recognised, from Boveri's well-known figures. Of course, the embryo is not supposed to be hermaphrodite; both sexes being included in one diagram merely for purposes of convenience. In the upper part of the diagram, attached to the ninety-sixth primary germ-cell, the course of oogenesis in the skate is shown. With the final division of the oogonium into two oocytes, o.c., the determination of sex is depicted as happening in the formation of male oocytes and female ones. These enter the period of growth, and then pass on to ripen.

Lower down, for comparison, the spermatogenesis of *Paludina*, a fresh water dioecious snail, with its two kinds of spermatozoa, is represented after the statements of Meves. The portions of the diagram appended to the ninety-sixth and thirty-second primary germ-cells, can naturally be applied to any of the remaining primary germ-cells, other than that which goes to form the embryo.

In the foregoing, what Weismann has termed the germinal track, nowhere touches the cells of the embryo. Neither, as we have seen, does it really lie within the asexual generation or phorozoon. It is along a line of unicellular organisms, which pass a portion of their life-cycle between one conjugation and the succeeding one within a sterilised individual, formed by the self-sacrifice of one for the good of the rest.

As revealed by the diagram, throughout this line of unicellular organisms, which are ever such, until one or other of them gets into the cul-de-sac of embryo-formation, there is a direct morphological continuity of germ-cells. This is all Nature demands: and this she accomplishes by the aid of unicellular organisms. All the observed phenomena of development, all those of heredity, are possible in this way. Notwithstanding apparent complexity, the process is simplicity itself, the

simplest kind of continuity conceivable. On the circle of life revolves the epicycle of the germ-cells. The circumference of the former is filled in by an interrupted succession of such epicycles. The constant sequence of these is the rhythm of reproduction, the gamut of life.

(To be continued.)

#### REFERENCES.

1. Weismann, A. "Vortraege ueber Descendenztheorie," vol. i. p. 449, 1902.
2. Weismann, A. *Op. cit.*, vol. i. p. 449.
3. Weismann, A. "Die Continuitaet des Keimplasmas," Jena, 1885, p. 44.
4. Noll, F., in Strassburger's "Lehrbuch der Botanik," 2te. Aufl., 1895, pp. 208-209.
5. Eckermann, "Gespraech mit Goethe," p. 117, Dec. 9, 1824.
6. Maupas, E. "Modes et formes de reproduction des Nematodes," in *Arch. Zool. exper.*, vol. viii. pp. 463-624, 11 pl., 1900.
7. Wilson, E. B. "The Cell-Lineage of *Nereis*," in *Journ. Morph.*, vol. vi. pp. 361-480, 1892. "Cell-Lineage and Ancestral Reminiscence," in *Wood's Holl Biol. Lectures*, 1898, pp. 21-42 (published in 1899).
8. *Mitt. Zool. Stat. Neapel*, vol. xiii. pp. 1-292, 1898.
9. Sars, M. "Ueber die Entwicklung der *Medusa aurita* und *Cyanea capillata*," in *Arch. Naturgesch.*, vol. vii., 1841.

### A CASE OF INTENSE AND GENERAL MUSCULAR ATROPHY CAUSED BY ENCEPHALOMYELITIS.<sup>1</sup>

By WILLIAM G. SPILLER, M.D.,

Associate Professor of Neurology and Professor of Neuropathology in the University of Pennsylvania.

From the Pennsylvania Training School for Feeble-Minded Children and from the William Pepper Laboratory of Clinical Medicine (Phœbe A. Hearst Foundation).

ABOUT a year ago I made a necropsy upon the body of a child in whom the muscular wasting was extreme and general. I have never seen a greater degree of muscular atrophy. The cause was found in encephalomyelitis. The inflammation of the central nervous system and the edema of the spinal cord were intense. The condition is not a very common one, and the case seemed worthy of study, especially as it resembled one that I recently

<sup>1</sup> Read before the Pathological Society of Philadelphia, Dec. 17th, 1903.



The photographs were taken after the death of the patient, and for them I am indebted to Dr W. G. Shallcross.



reported, in which a very similar group of symptoms was caused by unilateral internal hydrocephalus.<sup>1</sup>

The history as obtained from the case-books of the Training School is as follows:—

Margaret H. was born November 15th, 1886, and was the second child in the family. She was admitted to the Pennsylvania Training School for Feeble-Minded Children, July 13th, 1898.

The father, mother and child were born in England. Both parents are living and are in poor health. The father is a day labourer. He is a drunkard and feeble-minded, and probably has had syphilis. The paternal grandfather of M. H. died when forty-three years of age of phthisis; the maternal grandfather died of Bright's disease. Five brothers and one sister are now living, and are said to be in good health. The mother is an industrious woman but hysterical. M. was born at full term, in ordinary labour, and was nourished by the breast. She commenced to walk when she was two years of age. Epilepsy began when she was twelve years of age.

When admitted to the School in 1898, at the age of twelve years, M. was of feeble mentality, and had strabismus and slight ptosis of the left eyelid. The pupils were equal and the light reflex was preserved. The special senses could not be carefully tested because of her mental condition. She was not paralysed and her muscles were not spastic. She could speak, but said very little, and her speech was imperfect. The tendon reflexes were present. She was able to wash and dress herself.

A note was made that from May 15th to August 24th, 1901, M. was in the infirmary, was badly nourished and had spastic paralysis. Dr Llewellyn, one of the resident physicians, informs me that she saw M. almost every day during several years, and at no time did she show any symptoms of acute disease. There was so gradual a failure of physical and mental power that the difference in her condition was recognisable only after several weeks' observation. She died October 5th, 1902, at the age of sixteen years.

The necropsy was made October 8th. The measurements of the body were: From the top of the head to the great trochanter of the femur 69 cm., from the great trochanter to the

<sup>1</sup> Spiller, *The American Journal of the Medical Sciences*, July 1902.

external tuberosity of the femur 31 cm., from the external tuberosity to the sole of the foot 37 cm. The lower limbs were of the same length. There was slight equino-varus on each side, especially on the right side. The toes were in extension. The right leg was flexed at a right angle to the thigh and could not be extended. The left leg was flexed at an obtuse angle to the thigh, at an angle of about 120 degs. Both thighs were slightly flexed on the abdomen, the right more so than the left. The right leg at its middle portion measured in circumference 11 cm., the left leg measured 11.5 cm., the right thigh at its middle portion measured 14.5 cm., the left thigh measured 14.5 cm. The fingers of each hand were slightly flexed, but could be easily extended. No contracture was present at either wrist. The right forearm was flexed at a right angle to the arm, but could be extended a little. The left forearm was flexed at an angle of about 120 degs., but could also be extended a little. The right forearm in its middle portion measured in circumference 9.5 cm., the left forearm measured 9.5 cm., the right arm in its middle portion measured 9.5 cm., the left arm 9 cm. The entire body was intensely atrophied. The eyeballs were deeply sunken in the orbits, and the jaws had scarcely any muscle upon them. Deep grooves were at the inner borders of the scapulæ on account of the muscular atrophy. The skull where it was sawed through, 3 cm. above the supraorbital ridge, measured 1.2 cm., in the occipital region it measured 0.7 cm. Above the saw line the frontal bone was much thicker. The brain was very edematous and weighed 930 grammes. The spinal cord was unusually large, and even after it had been kept several weeks in hardening fluid it swelled out over the edges like a mushroom wherever it was cut. The swelling was caused by intense edema of the cord. An examination of the thoracic and abdominal viscera revealed no gross lesions.

The right and left paracentral lobules showed intense round cell infiltration about the vessels within the grey and white matter and within the pia. No hæmorrhages were found. The nerve cells of the cortex were much less numerous than normal, and were shrivelled and stained poorly. The alteration was the same in sections from the right occipital lobe. The round cell infiltration was very intense in sections from the optic thalamus, cerebellum and medulla oblongata, and was especially about



the blood-vessels. The pia over the medulla oblongata was only slightly infiltrated with cells, and its blood-vessels were not much thickened.

In sections from the cervical enlargement the white matter did not stain so sharply as in a normal spinal cord, and there was a homogeneous infiltration between the nerve-fibres. Some of the axis cylinders were swollen. Much cellular infiltration was found about the blood-vessels of the spinal cord, and to a less extent in the pia. The blood-vessels of the pia were not distinctly thickened. The anterior and posterior roots were nearly normal, both by the Weigert hematoxylin and the acid fuchsin stains, except that some axis cylinders were not distinct. The cellular infiltration about the blood-vessels was not so great as in the cerebral cortex. The crossed pyramidal tracts stained a little faintly by the Weigert hematoxylin method, and were probably slightly degenerated. No distinct degeneration was found in the cervical cord by the Marchi method. The nerve-cells of the anterior horns stained by the thionin modification of Nissl's method were intensely altered. The cells were pale, some were much vacuolated, most of them were much atrophied, and the chromophilic elements had almost disappeared. The blood-vessels of the cord were much congested.

The condition of the lumbar region was much like that of the cervical region, except that the perivascular cellular infiltration within the cord was less intense.

A piece of the left deltoid muscle examined was atrophied, but not nearly so much so as might be expected. The small nerves within the muscle stained well by the Weigert hematoxylin method. Micro-organisms were not found in stained sections.

The clinical history of this case is incomplete, but it is evident that the child was feeble-minded and epileptic, that spasticity, paralysis and atrophy were of gradual development, that the limbs gradually became contracted, that the child did not have any acute disease that could be regarded as the cause of the pathological changes, and that her father was a drunkard and weak-minded, and probably had had syphilis.

Extensive encephalomyelitis, as found in this case, is a well-recognised disease, but usually develops after some infection. The case which forms the subject of study in this paper was

remarkable because of the absence of any acute disease, because of the intensity and extent of the inflammation of the central nervous system, because of the intense edema of the spinal cord, and because of the general and extreme wasting of the muscles. It is strange that the child lived long enough for such atrophy to develop, inasmuch as the implication of the central nervous system was so widespread.

The lesions may have been syphilitic, and the symptoms do not make the diagnosis of hereditary syphilis improbable. I should hardly care to say positively that this was a case of cerebro-spinal syphilis, especially as the walls of the blood-vessels were not thickened to the extent usually found in cases of acquired syphilis. Hereditary syphilis may cause clinical signs appearing some years after birth.

Oppenheim,<sup>1</sup> who has made syphilis of the nervous system a subject of special study, says that hereditary syphilitic disease of the brain usually first manifests itself in the early period of life, but it is not uncommon to find the first signs at the age of puberty. It may first appear in the third or fourth decennium, as he has observed in two cases. The pathological alteration is the same as in acquired syphilis, only in the hereditary form it is frequently more severe and more extensive than in the acquired form, and causes developmental defects (atrophy, sclerosis, hydrocephalus, hypoplasia, etc.). The symptomatology is essentially the same as in acquired syphilis, only the mental defects are more pronounced and epilepsy is frequent. He refers to the implication of both brain and spinal cord in hereditary syphilis, and to cases reported by Jürgens, Siemerling, Böttiger and others.

Jordan,<sup>2</sup> of Heidelberg, has recently made a contribution to late hereditary syphilis. Hereditary syphilis, according to him, is usually manifest in the new-born child, or appears within the first or second week, or at furthest within the second or third month. It differs from the acquired syphilis of the adult only in the regularity and order of the symptoms. The periods are not clearly marked. Most children with hereditary syphilis die

<sup>1</sup> Oppenheim, *Lehrbuch der Nervenkrankheiten*, third edition, p. 835, and Nothnagel's *Specielle Pathologie und Therapie*, vol. ix. Part II. : "Die syphilitischen Erkrankungen des Gehirns."

<sup>2</sup> Jordan, *Münch. med. Wochenschrift*, No. 31, Aug. 4, 1903, p. 1324.

young; some may recover under specific treatment, and their later development may be normal, although recurrence of the symptoms is possible.

By late hereditary syphilis is meant the appearance of the signs of the disease after the fourth year—in the tenth, fifteenth, or twentieth year, or even later. This form may occur in a person who showed signs of syphilis in early childhood, or in one who showed no such signs. The tardy syphilis may appear any time between the tenth and twenty-fifth year, and often may be monosymptomatic. The late syphilis is usually confused with tuberculosis. Only a few cases of late hereditary syphilis have been reported, according to Jordan, and two cases are described by him.

### **LUMBAR PUNCTURE.**

C. MACFIE CAMPBELL, M.B., Ch.B.

THE withdrawal of cerebro-spinal fluid by lumbar puncture was introduced by Quinke of Kiel in 1890, and was at first looked upon as a valuable therapeutic agent in grave disorders of the central nervous system. The therapeutic value of the operation was soon found to have been over-estimated, while in certain cases the withdrawal of cerebro-spinal fluid relieves symptoms and occasionally has a curative effect, this measure would not have attained its present vogue were it not that Widal, Sicard, and Ravaut in 1900 called attention to its diagnostic value.

These authors, applying to the cerebro-spinal fluid the methods which Widal and Ravaut had fruitfully employed with regard to various other fluids of the body, showed that in certain pathological conditions the systematic examination of this fluid yields information of great diagnostic value which may materially modify the treatment.

The nature of the fluid in the various forms of acute meningitis was first investigated, and then the method was extended to most chronic nervous diseases of central origin. Neurologists recognising the value of the method have elaborated it considerably, devoting much attention to the consideration of its physical, chemical and cytological characters until the examination of the

cerebro-spinal fluid has become one of the current methods of neurological diagnosis.

The operation of tapping the subarachnoid space by lumbar puncture is extremely simple. The spinal cord with its pial envelope ends opposite the second lumbar vertebra, while the dura and arachnoid continue as far as the second sacral vertebra. There is therefore in this region a reservoir of cerebro-spinal fluid extending about 25 centimetres and measuring in breadth about 2 centimetres.

In this reservoir float the nerves of the cauda equina. Puncture in the third, fourth, or fifth lumbar interspace cannot touch the cord, while the nerve roots which are occasionally touched by the needle never suffer any damage. If the patient during the operation suddenly complains of a severe pain shooting down the leg the needle should be slightly withdrawn to disengage it; the fact of touching the nerve-root is no indication for discontinuing the operation.

The puncture is best made in the fourth lumbar interspace where the dura mater is most firmly attached (Sicard). There is thus less chance of the membrane being pushed onwards in front of the needle, and the needle thus failing to draw off fluid. To determine the fourth interspace one feels the highest point of the crest of the ilium on both sides: the line joining these points passes over the fourth interspace, and this point can be previously marked with silver nitrate if one be not familiar with the method.

The needle should be about 10 centimetres long and about  $\frac{5}{16}$  of a millimetre in bore for an adult—for a child a much smaller needle suffices. The best needle is made of platinum or platino-iridium which can be readily sterilised by heating over a spirit-lamp: where there is any risk of such a needle being broken, as with excited lunatics or with very muscular patients due to movement during the operation, a needle of steel should be used.

A small rubber tube is fixed to the needle so that the rate of outflow of the fluid may be easily regulated; while a wire to run through the needle if necessary should also be at hand.

The patient may either be sitting up leaning well forward or lying on his side; the latter position is preferable as in the upright position the fluid escapes more rapidly, there is more

tendency to a spasmodic contraction of the muscles, and there is greater difficulty in controlling an excited patient.

The patient lying on his side is brought to the edge of the bed, the head is raised by a pillow, the thighs well flexed on the pelvis; if the patient in this position bend his back as much as possible the vertebral laminæ are separated about  $1\frac{1}{2}$  centimetres.

The lumbar region is then carefully disinfected, the operator having again felt his landmarks, washes his hands, puts his left index on the fourth lumbar spine, and with the index of his right hand on the end of the needle enters it in the fourth interspace about half a centimetre from the middle line. The skin prick is not felt if the part be frozen with ethyl chloride, and usually the patient suffers little pain as the needle is steadily directed inwards with a slight inclination upwards and towards the middle line. As the needle comes on the ligament the guiding-finger experiences a characteristic sensation, and with a little firm pressure the needle is felt to penetrate a rather tough substance and the liquid begins to flow as soon as this resistance is overcome.

Should the needle in the course of the operation come against bone it must be slightly withdrawn and pushed in again slightly lower down until the ligament be found. The needle may be in position and yet no fluid come: the needle should be slightly rotated to disengage it from the obstruction, and if still no fluid come the wire should be passed along the needle to clear the point from a nerve or to remove a small clot which may have formed on the point during the passage through the deeper strata.

After a sufficient quantity of fluid has been withdrawn the needle is smartly withdrawn and collodion applied. The fluid should be allowed to flow of its own accord. Aspiration of the fluid should only be resorted to with the greatest care.

After the puncture the patient should rest for an hour or two in the horizontal position to obviate the occasional tendency to headache. If only 10 c.c. be withdrawn the patient suffers no inconvenience beyond perhaps a passing headache probably not due to the extraction of the fluid at the time of the operation, but to further leakage of the fluid through the hole in the dura.

In children one occasionally finds fairly severe vomiting caused by the puncture.

Normal cerebro-spinal fluid is clear as crystal. If the fluid that issues from the needle be bloody, the point is to be determined whether that be due to the operation or not. If it be pure blood that escapes, and if it show no sign of clearing up the puncture is of no use and must be repeated in another interspace: the escape of blood due to puncture of a venule need in no way disconcert the operator.

If the fluid be merely bloody there are various methods of finding out whether this be due to the operation or to the hæmorrhagic nature of the contents of the subarachnoid space.

If one receive the fluid in three different tubes and find that the tint of the three is the same, probably the blood is not due to the puncture: the tint may, however, be identical in the three tubes, and yet we may fail to demonstrate at the autopsy any hæmorrhage in the central nervous system (Sicard). Conversely, the tint may be successively lighter in the three tubes, and yet the colour may be due to a hæmorrhage, and not to the puncture. This is probably due to the gravitation of the effused blood towards the bottom of the subarachnoid *cul-de-sac* (Bard).

A second point which helps to distinguish between the two is that if the fluid be due to the puncture there is a clot formed at the bottom of the tube, which clot is not dissociated on shaking the tube, as it is where a hæmorrhage is the cause.

As a rule, Sicard found that fluid tinged with blood due to a hæmorrhage had a darker tint than where the blood was due to the operation.

If on centrifuging the sanguinolent fluid we find the hæmoglobin bands on spectroscopic examination there has been a hæmorrhage of the central nervous system; in some cases of meningitis, however, the cerebro-spinal fluid may take the blood due to the puncture and thus lead to a wrong diagnosis.

Bard has called attention to the occasional presence of an amber colouration of the liquid after centrifuging, the liquid, however, not giving the hæmoglobin reaction. The nature of the pigment is not quite certain; it may be the pigment of blood serum, or a transformation product of hæmoglobin under the action of the cerebro-spinal fluid.

The presence of this amber colouration makes very probable

the diagnosis of a hæmorrhage or of acute inflammation of the meninges. It is of surgical interest that a hæmorrhagic cerebro-spinal fluid may be caused by a mere contusion without fracture of the skull.

While this amber colouration renders a hæmorrhage probable, the absence of any colouration does not exclude hæmorrhage. In acute meningitis the presence of this pigment is accompanied by that of cellular elements, but in four cases of chronic meningitis quoted by Sicard no cellular elements accompanied the amber colouration: its importance in diagnosis is obvious from these cases.

The presence of a greenish or yellowish colouration has been noted by several authors in cases of chronic jaundice.

The accurate estimation of the density and pressure of the cerebro-spinal fluid has not yet been shown to have much clinical interest. The various instruments used to register the pressure give different results, and for clinical purposes it is sufficient to observe the method of exit of the fluid as the needle enters the subarachnoid space. The fluid may gush out with great force, as in some cases of uræmia, or it may issue drop by drop. The bore of the needle and its position in the canal, as well as the position of the patient, modify the method of exit of the fluid.

Of greater clinical interest is the estimation of the osmotic tension of the cerebro-spinal fluid. For its determination two methods may be employed—the cryoscopic and the hæmatolytic.

The cryoscopic method is more accurate than the latter, but is less suitable for general application. The freezing point of blood serum is  $-0.56^{\circ}$ , while that of normal cerebro-spinal fluid varies between  $-0.72^{\circ}$  and  $-0.78^{\circ}$  (Widal, Sicard and Ravaut), between  $-0.50^{\circ}$  and  $-0.60^{\circ}$  according to Achard, Lœper and Laubry. Taking the former estimate, the fluid has a greater molecular concentration than blood serum; in other words, is hypertonic with regard to it.

This hypertonicity is inverted in cases of acute meningitis where the cryoscopic point varies between  $-0.50^{\circ}$  and  $-0.56^{\circ}$ : here the cerebro-spinal fluid is hypotonic with regard to the blood.

It is always necessary to do the cryoscopic examination of the blood serum at the same time: in a case of pneumococcal cerebro-spinal meningitis the cryoscopic point of the fluid

was  $-0.59^{\circ}$ , but the blood serum gave the figure  $-0.71^{\circ}$ , showing that the normal relation was here inverted—a fact which the cryoscopic examination of the fluid alone did not bring out (Widal and Sicard).

This inversion if present confirms the diagnosis of meningitis, if absent does not exclude it, and in any case does not help to distinguish between the tubercular and the non-tubercular form.

In chronic nervous diseases the freezing-point is normal.

The hæmatolytic method of determining osmotic tension is much simpler if less accurate.

Normal cerebro-spinal fluid does not lake blood: laking only takes place after the addition to the fluid of about an equal quantity of distilled water. Where the osmotic tension becomes lowered, as in meningitis, a much smaller addition of water is sufficient to cause laking.

The method is simple, but hæmatolysis is a complex process, and depends upon the condition of the reds as well. Hæmatolytic power is not always a true measure of osmotic tension, and occasionally cryoscopy gives the opposite result to the hæmatolytic examination.

Wherever the symptoms suggest a meningitis the cerebro-spinal fluid should be bacteriologically examined. The results of such examination demonstrate often the existence of meningitis where the clinical symptoms leave us at fault.

Children in the infectious diseases often present meningeal symptoms which disappear, and are usually diagnosed as pseudo-meningitis.

Monod studying the spinal fluid in these cases has shown that the fluid may be serous, turbid or purulent, while the nervous centres show insignificant alterations. They are, therefore, true cases of meningitis, which are curable; in such cases the puncture has a therapeutic as well as a diagnostic value.

Similarly with regard to cases of tubercular meningitis, where the diagnosis is called in question merely owing to the recovery of the patient, Widal comes to the conclusion that unless inoculation of a guinea-pig with the cerebro-spinal fluid has proved negative, one has no right to deny the diagnosis.

A bacteriological examination may be made in three different ways.



There is the short method of centrifuging the fluid and staining the deposit ; but this method is hardly sufficient, and in a large minority of cases the bacillus of Koch escapes the direct microscopical investigation.

A culture on blood-agar is a more satisfactory method of demonstrating the bacillus, but the care required in following it, as well as the length of the process (14-21 days), make the method hardly suitable for clinical purposes.

The most certain method of determining whether the meningitis is of a tubercular nature or not is the intraperitoneal injection of 3 c.c. of the cerebro-spinal fluid into a guinea-pig. The method is simple, but requires three weeks.

The virulence of the cerebro-spinal fluid for the guinea-pig is found only in tubercular meningitis, and neither in non-tubercular meningitis nor in tubercular subjects affected with non-meningeal lesions, nor in acute syphilitic meningitis.

It is important to remember that the bacillus of Koch is not the only organism which may be present in tubercular meningitis ; it may be associated with other organisms. The demonstration of these organisms does not warrant us in concluding to the non-tubercular nature of the process.

The bacteriological examination enables us to distinguish the tubercular from the non-tubercular meningitis ; it enables us further to subdivide the latter.

The various forms of non-tubercular meningitis may be grouped into : (1) epidemic cerebro-spinal meningitis due to the pneumococcus of Talamon, the meningococcus of Weichselbaum, or the streptococcus of Bonome ; (2) the serous meningitis of Ziegler, caused by a variety of organisms ; among others, by pneumococcus ; (3) in this group one has demonstrated a large variety of organisms (Widal).

In other diseases of the central nervous system, including dementia paralytica, no organisms have been demonstrated.

The exact chemical constitution of cerebro-spinal fluid has not been determined, but most authors agree that normally there is a trace of serum globulin present, serum albumin being normally absent.

In boiling a normal cerebro-spinal fluid a slight opalescence and fine threads appear ; in certain pathological conditions the opalescence becomes exaggerated into turbidity and the threads become

more marked. One can by boiling estimate whether the fluid is pathologically albuminous or not. In cases of tabes, general paralysis and syphilitic meningo-myelitis and hemiplegia, Widal and Sicard found always this pathological reaction; and Guillain, in a series of sixteen cases of general paralysis, found this pathological reaction with presence of serum albumin; Léri, however, arrives at different results.

The chemical test is important because it is not always parallel with the cytological; in cases of chronic meningitis the two reactions are usually parallel, but Monod has shown that in the acute meningitis of children the pathological albumin reaction may be present with absence of cells.

A similar condition has been found in erysipelas of the face, polymorphous erythema, cerebral hæmorrhage, secondary syphilis (Widal, Sicard and Ravaut).

In acute meningitis fibrin is present, although absent from the normal fluid. Apart then from all elaborate methods, we can say that every cerebro-spinal fluid, although quite clear in which a coagulum of fibrin forms, is pathological and probably due to an acute meningitis (Widal and Sicard).

It is unnecessary to do more than mention the question of meningeal permeability, which is of little practical interest. Normally the meningeal covering of the brain and cord does not allow potassium iodide, even although taken in large quantities, to pass through into the cerebro-spinal fluid. In certain conditions we find that this restraining action does not exist, as, for example, in cases of tubercular meningitis.

Of all the methods used in studying the cerebro-spinal fluid none has been of such value to the clinician as the examination of the cellular elements contained in it. In the three years which have elapsed since Widal, Sicard and Ravaut published their article on the cytological diagnosis of tubercular meningitis, a large number of nervous diseases have been investigated from this point of view, and the value of the method as giving accuracy and certainty to a diagnosis is well established.

Thanks to the examination of the fluid we can make a diagnosis much more precise than the mere examination of the clinical symptoms would allow. The method does not allow one to grind out a mechanical diagnosis nor absolve one from an accurate study of the clinical phenomena; it is only in con-

junction with the latter that the result of microscopical examination can be correctly appreciated. Normally the cerebro-spinal fluid is almost free from elements, containing no polymorphonuclear leucocytes, and few or no lymphocytes. Where there is meningeal irritation cellular elements begin to appear in the cerebro-spinal fluid.

In order to demonstrate the elements present one must rigorously follow the technique insisted on by Widal. In order to compare the results of different punctures the amount of fluid withdrawn should always be the same—between 3 and 6 c.c. This amount of fluid is received in the tube in which it is to be centrifuged, the tube tapering somewhat to the bottom. After centrifuging for ten minutes the fluid is decanted and the tube is left to drip on to a piece of blotting paper until it is completely drained. Holding the tube still reversed one scrapes the bottom on which the elements have been deposited with a very fine capillary pipette: the minute quantity of liquid remaining in the tube rises into the pipette, carrying the elements with it. If the fluid rise more than 2 c.c. in the pipette the tube has not been left to drip long enough.

The fluid is then blown carefully on to three slides—each of the three droplets covering not more than two or three square millimetres on the slide. The slide is then dried in the air or in the oven at a low temperature, and is then fixed in a mixture of equal parts of alcohol and ether for twenty minutes: the fixative must not be too roughly poured on, otherwise the crystals of sodium chloride formed as the fluid dries may be washed away and carry with them the cells. The preparations are stained with Unna's polychrome blue (ten minutes), haematoxylin and eosin, and Ehrlich's triacid (after heat).

In a preparation so made from a normal cerebro-spinal fluid one never sees more than two or three lymphocytes in one field under the oil immersion.

In cases of meningitis, tubercular and non-tubercular, and in various chronic nervous diseases the microscopical examination shows a field with numerous elements.

In tubercular meningitis the cells are almost entirely lymphocytes. Often these are mixed with a few polymorphs and some large mononuclear elements, and in some cases the polymorphs may nearly equal in number the lymphocytes (Widal).

Some authors cite cases where there was even a leucocytosis. Before concluding that there is a leucocytosis a differential count of 500 elements is necessary.

In some of these cases of leucocytosis it was possible to exclude secondary infection. According to Concetti, who in these cases found a large number of bacilli in the fluid, the leucocytosis is due to the irritation of the bacillus, the lymphocytosis to the irritation of the toxin.

The general rule, however, is that in tubercular meningitis one finds a lymphocytosis of the cerebro-spinal fluid. One must, however, keep in mind the following points:—1. A lymphocytosis is also found during recovery from an acute non-tubercular meningitis: the evolution of the disease prevents any misinterpretation of the facts. 2. As mentioned above, one finds occasionally a leucocytosis in tubercular meningitis. 3. Exceedingly rarely no elements are found in tubercular meningitis: perhaps this is due to the occlusion of the foramen of Magendie. The occasions are so rare that the value of the sign is little impaired, and where in a doubtful case of meningitis no elements are found one can rule out this diagnosis. The cytological examination is particularly useful in diagnosing meningitis from meningism, or syndrome of Dupré consisting of cerebral troubles of functional or toxic origin occurring, *e.g.* in typhoid fever, pneumonia, rheumatism, etc. 4. One has a lymphocytosis in various chronic diseases of the nervous system, and also exceptional lymphocytosis has been found in tumour of cerebellum, fracture of the base of brain, acute syphilitic meningitis, typhoid meningitis, pneumonia with delirium, and occasionally in simple excess of tension of the fluid.

In acute non-tubercular meningitis there is usually a marked leucocytosis, sometimes rendering the cerebro-spinal fluid purulent. In the chronic nervous diseases one gets cellular elements in the fluid or not, according as the meninges are irritated or not.

In general paralysis a lymphocytosis of the cerebro-spinal fluid is one of the earliest signs, and is practically constant: its absence, however, is noted by Achard and Grenet, Joffroy and Mercier, Ballet and Delherm, but is so rare that the method loses little of its value. What the pathological explanation of this absence of lymphocytosis is in cases where the pia

mater of the brain is undeniably implicated is not yet clear. The assumption of a very slight implication of the pia does not solve the difficulty, for Nageotte has shown that lymphocytes appear after a minimal irritation of the pia.

In various other forms of mental disease investigated, true psychoses and dementias of different origin, lymphocytosis has been absent (Dupré and Devaux).

This is also the case in hysteria, neurasthenia and epilepsy. The cytological examination, however, does not enable one to diagnose between a general paralytic and a chronic alcoholic whose meninges are affected. Thus Dufour has seen the lymphocytosis develop in the case of an alcoholic, where the absence at first of lymphocytes had enabled the diagnosis from general paralysis to be established.

In tabes dorsalis, lymphocytosis is the almost universal rule, the degree being greater the nearer the puncture is to the beginning of the disease. Here again we must acknowledge the very occasional absence of lymphocytes.

Armand-Delille and Camus quote a series of thirteen tabetics in only four of whom there was a distinct lymphocytosis. These figures created much astonishment and the criticism of Widal with the statistics produced in answer to their communication make it certain that their technique had not been sufficiently rigorous.

This lymphocytosis in tabes is of great interest with regard to the pathogeny of the disease, and is intelligible with Marie's formulation of the process as a histolymphangitis of the posterior lymphatic system of the cord of syphilitic nature.

In both tabes and general paralysis, lymphocytosis is one of the earliest and most constant symptoms, and its determination in suspected cases should never be omitted: its presence at an early stage is the indication for an active mercurial treatment which may do much to check the progress of the disease.

Bahinski was the first to insist upon the importance of the sign of Argyll Robertson, as showing a syphilitic affection of the nervous centres, especially of the meninges. This sign reveals an incipient tabes, and with it is associated a marked lymphocytosis of the cerebro-spinal fluid.

Widal and Ravaut have especially studied the fluid in secondary and tertiary syphilis. If one find even a slight

lymphocytosis in a syphilitic, a thorough examination of the nervous system must be made, leading often to the detection of some nervous symptoms (Widal). Lymphocytosis is the rule in syphilitic meningomyelitis and if found in a case of cerebral hemiplegia a certain time after the shock, it almost certainly points to a syphilitic origin.

The headache which is such a constant symptom in secondary syphilis is in certain cases associated with a lymphocytosis of the cerebro-spinal fluid: thus in eight cases examined by Milian, Crouzon and Paris, two presented in the fluid numerous elements, chiefly lymphocytes. These authors distinguish among syphilitic headaches a meningeal form characterised by its intensity, and by the excess of tension and presence of elements in the cerebro-spinal fluid. No other sign of the affection of the nervous system was to be traced in these patients.

Secondary syphilitics who show distinct signs of meningeal reaction have usually intense cutaneous manifestations (Ravaut): this simultaneous affection of skin and membranes shows the partiality of the syphilitic virus for the ectoderm (Brissaud).

This relation can only be revealed by lumbar puncture, for in his cases Ravaut was unable to trace any clinical symptom of a nervous lesion, save occasionally the headache.

In two cases of facial paralysis of syphilitic origin investigated by the same author there was a marked lymphocytosis: he also records the fact that the headache and vomiting occasionally caused by the puncture were more frequently met with in cases where there was found a marked cellular reaction.

In a short sketch of the results of lumbar puncture as a method of clinical examination, it is impossible to go fully into the various diseases which have been investigated. Mention may, however, be made of the interesting results in various forms of herpes zoster where the cytological examination suggests a division of zona into three groups — metameric, ganglioradicular and peripheral (Widal).

It is impossible to do more than mention the therapeutic applications of lumbar puncture: in certain forms of meningitis, in uræmic conditions, even with the fluid not under high pressure, in the headache of secondary syphilis the withdrawal of the fluid causes great relief. One has also used lumbar puncture as a method of applying various drugs, especially cocaine as an

anæsthetic: many of the drawbacks to this latter have been removed by Sicard's discovery that the cocaine if introduced in a solution of the patient's own cerebro-spinal fluid no longer produces the same meningeal reaction.

In conclusion, the following points may be insisted upon:

Lumbar puncture is a simple and innocuous procedure: sometimes it is in itself of therapeutic value.

It renders great service to the clinician by establishing a diagnosis between organic and functional affections, and by giving a precision which the clinical phenomena fail to give.

It enables one to diagnose certain diseases in their earliest stages and thus to employ therapeutic measures under the most favourable conditions.

#### BIBLIOGRAPHY.

*Achard and Loeper.*

Sur la perméabilité pie-mérienne. *La Presse méd.* No. 73, 1901.

*Armand-Delille and Camus.*

Examen cytologique du liquide céphalo-rachidien dans le tabes. *Rev. Neur.*, 28th Feb. 1903. (With discussion by Babinski, Déjerine, Dufour.)

*Bard.*

Résultats cliniques de l'appréciation de la tonicité du liquide céphalo-rachidien par son action sur les globules rouges du porteur. *Soc. de Biol.*, 16th Feb. 1901.

Des variations pathologiques du pouvoir hémolytique du liquide céphalo-rachidien. *Semaine méd.*, 14th Jan. 1903.

Des colorations du liquide céphalo-rachidien, d'origine hémorrhagique. *Semaine méd.*, 14th Oct. 1903.

*Belletre.*

La ponction lombaire chez les syphilitiques. Cytologie du liquide céphalo-rachidien. *Thèse de Paris*, 1902.

*Bellisari.*

Toxicité du liquide céphalo-rachidien dans la paralysie générale.

*Riforma Med.*, 1899.

*Bernheim and Moser.*

Ueber die diagnostische Bedeutung der Lumbalpunktion. *Wiener klin. Woch.*

*Berthet.*

La Rachicocainisation. *Thèse de Paris*, 1902.

*Blavot.*

Traitement des méningites cérébro-spinales par la ponction lombaire. *Thèse de Paris*, 1902.

**Bernasconi.**

Sur la valeur diagnostique, pronostique et thérapeutique de la ponction lombaire dans les fractures du crâne et du rachis. *Thèse de Lyon*, 1902.

**Boscolo.**

Meningism and lumbar puncture. *Rivista di Clin. med.*, 31st March 1900.

**Carrière.**

Examen cytoscopique du liquide céphalo-rachidien dans la sclérose en plaques. *Soc. de Biol.*, 23rd March 1901.

**Cassel.**

Lumbalpunktion für Kinder. *Jahrb. f. Kind.*, No. 47, 1898.

**Dechy.**

Le signe d'Argyll Robertson et la cytologie du liquide céphalo-rachidien. *Thèse de Paris*, 1902.

**Duflos.**

La ponction lombaire en psychiatrie. *Thèse de Paris*, 1901.

**Faisans.**

Cyodiagnostic de la méningite tuberculeuse. *Soc. med. des hôp.*, 4th July 1901.

**Gilbert and Castaigne.**

Le liquide céphalo-rachidien dans la cholémie. *Soc. de Biol.*, 27th Oct. 1900.

**Devaux.**

La ponction lombaire et le cyodiagnostic. *Centralbl. für Nervenhe. u. Psychiat.*, No. 161, 1903.

**Guillain and Parant.**

Sur la présence d'albumines coagulables par la chaleur dans le liquide céphalo-rachidien des paralytiques spinaux. *Rev. Neur.*, 30th April 1903.

**Joffroy and Mercier.**

De l'utilité de la ponction lombaire pour le diagnostic de la paralysie générale. *Congrès des méd. alién. et neurol.*, Grenoble, Aug. 1902.

**Lutier.**

Les nouveaux procédés d'investigation dans le diagnostic des méningites tuberculeuses. *Thèse de Paris*, 1903. With a full bibliography.

**Joffroy.**

Contribution à l'étude cytologique du liquide céphalo-rachidien. *Annales méd.-psych.*, Sept. 1901.

**Labbé and Castaigne.**

Examen du liquide céphalo-rachidien dans deux cas de méningites cérébro-spinales terminées par la guérison. *Soc. med. des hôp. de Paris*, 4th April 1901.

**De Lapersonne et Le Sourd.**

Le liquide céphalo-rachidien chez les syphilitiques. *Soc. de Biol.*, 10th January 1903.



*Maillard.*

De la valeur clinique du cytodagnostic céphalo-rachidien dans les cas douteux de paralysie générale progressive. *Thèse de Bordeaux*, 1901.

*M<sup>r</sup> Vail.*

Spinal puncture in "Uræmia." *Brit. Med. Journ.*, p. 1054, 24th October 1903.

*Milian.*

Le liquide céphalo-rachidien hémorrhagique. *Gaz. hebdom. de méd. et de chir.*, 7th Aug. 1902.

*Milian and Legros.*

Le liquide céphalo-rachidien dans le tétanos spontané. *Soc. de Biol.*, 30th March 1901.

*Milian, Crouzon and Paris.*

La cephalée syphilitique éclairée par la ponction lombaire. *Soc. med. des hôp. de Paris*, 14th Feb. 1902.

*Nawratzki and Arndt.*

On variations of pressure in the cerebro-spinal fluid in convulsive attacks. *Allg. Zeits. f. Psych.*, Oct. 1899.

*Orefici.*

The passage of iodine and bromine into the cerebro-spinal fluid of children. *Sixth Italian Congress of Pediatrics*. Florence, Oct. 1901.

*Pellagot.*

Résultats obtenus par la ponction lombaire. *Thèse de Paris*, 1902.

*Percheron.*

Du diagnostic de la méningite tuberculeuse chez l'enfant. Valeur de la ponction lombaire. *Thèse de Paris*, 1903. With full bibliography.

*Pinauk.*

Formes frustes de la méningite cérébro-spinale dite épidémique. Cytodagnostic du liquide céphalo-rachidien. *Thèse de Paris*, 1901.

*Quincke.*

Ueber Lumbalpunktion. *Die deutsche Klinik am Eingang des zwanzigsten Jahrhunderts*, vol. vi.

*Ravaut.*

Étude cytologique du liquide céphalo-rachidien chez les syphilitiques. *Ann. de Dermat. et de Syph.*, Jan. 1903.

Le liquide céphalo-rachidien des syphilitiques en période secondaire. *Ann. de Derm. et de Syph.*, July 1903. With bibliography.

*Ravaut and Darré.*

Contribution à l'étude des herpès génitaux. Étude du liquide céphalo-rachidien. *Gaz. des hôp.*, Oct. 15, 1903.

*Ravaut and Aubourg.*

Le liquide céphalo-rachidien après la rachicocainisation. *Soc. de Biol.*, June 15, 1901.

*Raymond and Sicard.*

Le liquide céphalo-rachidien dans un cas d'hydrargyrisme chronique. *Soc. de Neur.*, May 15, 1902.

*Sainton et J. Ferrand.*

Fracture du crâne et méningite cérébro-spinale. Contribution à l'étude du liquide céphalo-rachidien hémorrhagique. *Gaz. hebdl.*, Dec. 5, 1901.

*Schæfer.*

The cerebro-spinal fluid in some mental affections. *Arch. f. Psych.*, 1902, fasc. 2.

*Sicard.*

De la forme ambulatoire des méningites bactériennes. Cytodiagnostic du liquide céphalo-rachidien. *Presse méd.*, Aug. 21, 1901.

Le liquide céphalo-rachidien. Ponction lombaire et cavité sous-arachnoidienne, pp. 192, published by Masson et Cie.

Chromodiagnostic du liquide céphalo-rachidien. Hémorrhagies du névraxe. Méningitis. Ictères. *Presse méd.*, Jan. 25, 1902.

*Sicard and Brecy.*

Méningite cérébro-spinale ambulatoire curable. Cytologie du liquide céphalo-rachidien. *Soc. méd. des hôp.*, April 25, 1901.

*Widal.*

Cytologie du liquide céphalo-rachidien des syphilitiques. *Soc. méd. des hôp. de Paris*, June 27, 1901.

*Widal, Sicard and Ravaut.*

Cytodiagnostic de la méningite tuberculeuse. *Soc. de Biol.*, Oct. 13, 1900.

Présence d'un pigment dérivé dans le liquide céphalo-rachidien au cours des ictères chroniques. *Bull. de la Soc. de Biol.*, Feb. 8, 1902.

Cryoscopie du liquide céphalo-rachidien. *Soc. de Biol.*, Oct. 20, 1900.

*Widal and Ravaut, Widal and Sicard.*

In Bouchard's "Traité de pathologie générale," vol. vi.

*Widal, Sicard and Ravaut.*

A propos du cytodagnostic du tabes. With discussion and communications by other authors. *Rev. Neur.*, 31st March 1903.

In addition to the above mentioned articles one can consult the numerous discussions on communications made to the Société médicale des hôpitaux de Paris (Babinski and Nageotte, Nageotte, Joffroy, Dupré and Devaux, Séglias and Nageotte, Widal and Le Sourd, Achard, Chauffard, Crouzon, Brissaud, etc.) during the years 1901-1903: these are published in the weekly "Bulletins et Memoires" of the society.

---

# Abstracts

## ANATOMY.

### THE SO-CALLED "AFFENSPALTE" IN THE HUMAN

- (1) (EGYPTIAN) BRAIN. G. ELLIOT SMITH, *Anatom. Anzeiger*. Bd. xxiv., No. 2/3, October 31, 1903, p. 74.

THIS is a preliminary note calling attention to the fact that a characteristic and undoubted occipital operculum, and an "Affenspalte" resembling that of the gorilla, often occur in the Egyptian brain, and probably also in those of Europeans and other races. The term "Affenspalte" and its equivalents ("external perpendicular" and "external parieto-occipital" sulci) are so obviously inappropriate, and have been employed with so many different meanings, that the new term "sulcus occipitalis lunatus" has been coined to signify the definite sulcus, the overgrown caudal lip of which forms the occipital operculum.

Definite representatives have also been found in the human brain of the sulcus occipitalis superior and sulcus occipitalis inferior of the apes. The term sulcus occipitalis paramesialis has been applied to a furrow which is often found in the gibbon's and the gorilla's brains skirting the dorsal edge of the hemisphere behind the parieto-occipital sulcus. This furrow is almost always present in the human brain, in which, however, it often becomes swept on to the mesial surface.

This short note is merely the preliminary announcement of the results of an extended investigation (now in course of publication) to demonstrate the homologies and range of variation of these sulci in a large series of simian and human brains. In the complete work it will be shown that the relation of the stripe of Gennari to the superior, inferior and lunate occipital sulci confirms the correctness of their identification, and points to the fact that most of the "area striata" (or Gennari's stripe-bearing cortex) has slipped backward on to the mesial surface in the human cerebral hemisphere.

AUTHOR'S ABSTRACT.

### ON A HITHERTO UNDESCRIBED NUCLEUS LATERAL TO THE

- (2) FASCICULUS SOLITARIUS. E. LINDON MELLUS, *Am. Journ. of Anat.*, Vol. ii., No. 3, 1903, p. 361.

THE author draws attention to a group of large, round, oval, or pear-shaped cells in the medulla of the dog, which lie lateral to the fasciculus solitarius. These cells, which vary in number from one to eight in successive sections, stain more deeply with carmine

than any of the cells in their immediate neighbourhood. They are larger than the adjacent cells of the eighth nucleus, and are separated by a distinct interval from Deiter's nucleus. The author refers to a clump of cells in the human medulla which occupy a very similar position. In the human subject the cells are much more numerous though smaller in size.

EDWIN BRAMWELL.

**ON THE STRUCTURE OF THE NERVE CELLS. (WHAT ARE**

- (3) **NISSL'S BODIES?**) C. CHENZINSKI, *Neurol. Centralbl.*, Nov. 16, 1903, p. 1045.

THE author investigated the structure of the anterior cornual cells of the cord in the human subject and in some vertebrates. The tissue was hardened in 5 per cent. formalin, cut in longitudinal sections of varying thickness (5-10 m.), and stained with methylene blue, thionin, and toluidin blue.

In longitudinal sections of the cord of the ox the whole protoplasm was filled with coloured strands running into all parts of the cell, into the protoplasmic processes, and from one to another of these across the cell body. The strands passing from one process to another were curved with the convexity towards the nucleus, others again running from one pole of the cell to the opposite pole were bent with the concavity looking towards the nucleus. Generally, however, the strands lay parallel to each other and pursued a wavy course.

In the human subject the strands more often appeared in the form of chains, consisting of short rods or spindles with unstained substance between their ends. In the protoplasmic processes, however, unbroken strands occurred just as frequently as the chain forms.

Chenzinski states definitely that the above described fibres are perfectly distinct from the fibrils of Bethe and Apáthy, and should the cell be cut in transverse section then they appear as granules or flakes. He therefore concludes that the Nissl granules are nothing more than transverse sections of longitudinal bands of chromophile material.

DAVID ORR.

**THE DIRECT PYRAMIDAL TRACT AND THE "CRESCENT"**

- (4) **PYRAMIDAL TRACT.** MARIE et GUILLAIN, *Semaine méd.*, Jan. 21, 1903.

**THE HOMOLATERAL PYRAMIDAL TRACT.** MARIE et GUILLAIN,

- (5) *Rév. de méd.*, Oct. 10, 1903.

In the first of these papers the authors seek to explain the frequent variations in the size, shape and position of the direct

pyramidal tract (bundle of Türk) by a difference in the site of the original lesion. From a detailed examination of more than twenty-five cases of degeneration involving this part of the spinal cord, they come to the following conclusions: If the primary lesion is cerebral, there is found usually a small area of sclerosis, which in the cervical region occupies the internal and posterior part of the anterior column, close to the anterior median fissure. Posteriorly it touches the anterior commissure; anteriorly it never extends further than half-way between this commissure and the anterior surface of the cord. It may be quadrilateral, oval, or elliptic in shape. Its average width is one millimetre, or a millimetre and a half. It diminishes in size in the lower cervical region, and seems to have disappeared in the upper dorsal. But the authors have often found below this a slight *atrophy* (no sclerosis) of the anterior column. With Marchi's method some degeneration is usually found in the lumbar or even the sacral cord. If, on the contrary, the primary lesion is in the pons or cerebral peduncle, the degeneration in the anterior column is much more extensive. It occupies about a third or a half of the column, and is prolonged outwards along the anterior surface of the cord. It is proposed to call this tract the "crescent tract" (*faisceau en croissant*). This special degeneration is easily followed to the lumbar region, and by the method of Marchi to the sacral.

The authors suppose that the fibres of the crescent bundle arise somewhere from the innumerable cells one finds in the peduncle in the sub-optic region, and in the pons in the neighbourhood of the pyramidal path. Thus they may fairly be termed para-pyramidal fibres.

In the second paper three questions are submitted:—

1. Is the non-paralysed side affected in hemiplegia?
2. Are the pyramidal fibres degenerated in both lateral columns in hemiplegia?
3. If a bilateral lesion exists, does it explain the clinical phenomena?

In the adult, it is rare to find the non-paralysed side affected; when it is, one ought always to think of a double hemiplegia incomplete on one side; for these incomplete hemiplegias are found with very great frequency in old patients with arterio-sclerosis, in whom foci of disintegration (in the basal ganglia) are usually present in both hemispheres.

With the method of Marchi one finds constantly a very small and scattered group of degenerated fibres in the lateral column opposite to the main lesion. With the method of Weigert there is never any sclerosis. If there is, the primary lesion is bilateral. The authors believe that these few fibres form a real and constant homolateral pyramidal tract.

But their degeneration does not explain the troubles on the sound side in hemiplegia. They are too few in number. Diminution in muscular force, exaggeration of reflexes and ankle clonus on the non-paralysed side, and post-mortem considerable sclerosis of the pyramidal tract on that side, can only be explained on the supposition that the primary lesion was a bilateral, not a unilateral one.

S. A. KINNIER WILSON.

# RESEARCHES ON THE ORIGIN AND INTRACEREBRAL

## (6) COURSE OF THE MOTOR NERVES BY MEANS OF THE METHOD OF INDIRECT WALLERIAN DEGENERATION.

A. VAN GEHUCHTEN. *Névraze*, vol. v., f. 3, 1903, p. 265.

In 1898 the author published a series of observations upon the grouping of the cells in the nuclei of the third, fourth, sixth, seventh, ninth and tenth cranial nerves. He adopted the method of dividing the individual nerves and then studying the distribution of the chromolytic changes. Contrary to the experience of other observers, van Gehuchten finds that "*la section d'un nerf spinal est toujours restée, entre nos mains, sans effet aucun sur les cellules radiculaires correspondantes.*" In a large number of rabbits he has *ruptured* the sciatic nerve and has never observed chromolysis in the anterior horns of the lumbo-sacral cord. For a number of years he has been engaged in studying the origin of the nerve of Willis. Although he has *divided* this nerve within the spinal canal on the surface of the spinal cord, he has never seen any chromolysis in the grey matter of the corresponding part of the spinal cord.

A disadvantage of the method of Nissl is that the nerve fibres cannot be traced from the seat of the lesion to the cells from which they arise.

Again, there is another fallacy which requires consideration. We have no means of proving that the chromolytic changes are confined to those cells the axis cylinders of which have been injured.

In this research van Gehuchten has employed the method of retrograde degeneration or, as he prefers to term it, *indirect Wallerian degeneration*. He has shown that when a peripheral motor nerve is *torn out* a rapid atrophy takes place in the cells from which it arises, and a consecutive degeneration of the central part of the fibre. He holds that this degeneration is not ascending or retrograde, but that it is descending or cellulifugal, and occurs in consequence of the injury to the cell.

The author has used extensively this method of tearing out (*arrachement*) individual nerves and then tracing their intracerebral course by the Marchi method. He has at the same time

employed the method of Nissl. Two animals must be used for each observation. One should be killed after eight or ten days, and the corresponding part of the nervous system examined by Nissl's method in order to show the chromolytic changes. The other animal should be kept alive for from 30 to 40 days, the nervous tissues treated by Marchi's method, and the intracerebral course of the fibres, which will have undergone indirect Wallerian degeneration, traced.

Space will only permit of the briefest reference to the results obtained.

After enucleating the eyeball all the soft parts were torn out of the orbital cavity and the intracerebral course of the ocular nerves studied.

Observations on the third nerve confirmed the previous results of the author and Biervliet. Some of the fibres come from the opposite side, their cells of origin lying in the dorsal part of the inferior three-fifths of the common grey mass. For details as to the exact origin of these fibres the original paper must be consulted.

The fourth nerve is formed chiefly of crossed fibres, although a certain number of fibres come from the same side.

All the fibres of the sixth nerve are direct; in other words, they arise from cells on the same side of the middle line.

The seventh nerve is also formed entirely of uncrossed fibres.

Both the motor and sensory fibres of the tenth nerve are uncrossed. There are two distinct nuclei, a ventral nucleus which forms the major part of the nucleus ambiguus and a dorsal nucleus which belongs entirely to the tenth nerve.

The nerve of Willis is a spinal nerve formed exclusively of uncrossed fibres. It rises from the first five cervical segments.

The hypoglossal nerve is also composed entirely of direct (uncrossed) fibres which all arise from the nucleus in the medulla first described by Stilling.

Applying the method of indirect Wallerian degeneration to the 8th cervical and first dorsal spinal nerves, van Gehuchten concludes that the motor fibres which they contain are exclusively direct, an arrangement which he thinks probably holds good for the anterior root fibres of all the spinal nerves.

EDWIN BRAMWELL.

**THE SILVER-IMPREGNATION OF NEUROFIBRILS. MAX  
(7) BIELSCHOWSKY, *Neurolog. Centralbl.*, 1903, N. 21, p. 997.**

**THE method is as follows:—**

1. Harden the organs, which should not be taken earlier than twenty-four hours after death, in 12 per cent. formol.

2. Cut by the freezing microtome. The sections must not be over 20  $\mu$  thick. They may be preserved in 1 per cent. formol.

3. Soak the sections in a 2 per cent. solution of  $\text{AgNO}_3$  in distilled water for 12 to 24 hours.

4. Place for 10 to 20 seconds in 3 per cent. solution of ammonia, in which the sections will take a yellow colour, the silver nitrate being transformed into silver-diammoniumnitrate.

5. Put the sections for 10 minutes in a 20 per cent. solution of formol, to which may be added one drop of a concentrated solution of carbonate of lithium for each 10 c.c. of water.

6. Pass through 3 per cent. solution of ammonia.

7. Transfer directly to  $\frac{1}{2}$  per cent. solution of nitrate of silver in distilled water. Keep the sections moving with a glass needle, until they assume a brownish colour (half minute). Only a few sections can be treated without filtering or renewing the fluid on account of deposits of metallic silver.

8. Let the sections remain in 20 per cent. formol until their colour is dark brown. (In impregnation of cortex this part of the process may advantageously be omitted.)

9. Pass again through 3 per cent. solution of ammonia, in which the colour of the sections will change to brown-black (formation of aldehyde bases).

10. Transfer to 20 per cent. formol for a few minutes, or, if the colour is very dark, to distilled water.

11. Tone the sections in an acid gold bath (2 to 3 drops of 1 per cent. gold chloride in 10 c.c. water, to which are added 2 to 3 drops of glacial acetic acid), until they appear grey or grey violet. Platinum toning gives less contrast.

12. Fix for a few seconds in an ordinary acid fixing bath diluted with 20 to 30 times the quantity of water.

13. Mount as usual (wash in water, then spirit, absolute alcohol, carbol-xylol, Canada balsam).

By this method the intracellular fibrils, medullated and non-medullated nerve fibres and Golgi-nets are stained. The aspect of the intracellular fibrils is the same as by the method of Bethe. On the other hand the pericellular net appears as a dense felting or fenestrated membrane, not as the narrow-meshed network enveloping the cell and dendrites "as trousers," seen in Bethe-preparations. In examining the impregnated cells, the question arises if there is any essential morphological difference in the structure of dendrites and axons. The collaterals are shown to be more numerous in the newly-born than in adults.

A fault of the method is that it frequently results in impregnation of fibrillar connective tissue, glia fibres, and even tigroid bodies of nerve cells, as well as of neurofibrils.

HANS EVENSEN.



## PATHOLOGY.

**NOTE ON THE PATHOLOGICAL ANATOMY OF ACUTE DIFFUSE**

(8) **MYELITIS (MYELITIS WITH EPITHELOID CELLS).** WEILL and GALLAVARDIN, *Rev. Neurol.*, Oct. 31, 1903.

THIS is the description of the histology of a case, the clinical account of which was published in *Lyon Méd.* for Aug. 9, 1903. The latter is briefly abstracted thus: Paraplegia of insidious onset, without obvious cause, which became complete in eight to ten days, and then presented a picture of a complete transverse lesion of the cord. A month and a half later bilateral amaurosis appeared and became complete in a few days, but later improved slightly. Death resulted from broncho-pneumonia. At the autopsy very intense diffuse myelitis was found involving the lower dorsal and lumbar regions, together with double optic neuritis, slight peripheral neuritis and diffuse interstitial encephalitis.

On microscopical examination at the level of the softened areas the anterior horns, anterior and lateral columns, and the anterior and posterior roots were infiltrated with inflammatory small round cells. The meninges were slightly thickened but contained few cellular elements, and the large perimedullary vessels were but slightly affected. The most severe lesion was situated in the posterior columns, where the mesial septum was destroyed by the formation of a large central cavity. Around the cavity there were neither inflammatory cells nor occlusion of vessels, but the tissues were densely packed with large, clear, non-granular cells, polyhedral in shape from mutual pressure, and possessing small, round, darkly staining nuclei, often double, which occupied scarcely one-tenth or one-twentieth of the bulk of the cell. The central cavity was almost filled with similar cells, but here the cell body contained fat granules and was spherical in shape. Where the tissues were less damaged similar epitheloid cells were arranged in layers one to three deep around the vessels, and where this sheath was but one layer deep the appearance presented was very similar to that of a renal tubule. Apart from the vessels, islets of these cells were to be seen lying among the nerve tissues. In other places, especially at the posterior border of the posterior columns, islets were found, the central cells of which were indistinguishable from ordinary inflammatory cells, but which, as the periphery of the group was approached, took on more and more cell body until they came to resemble very closely the epitheloid cells described above.

References to similar cases in the literature and a short discussion as to the origin of these epitheloid cells follow, and the authors express their opinion that they are derived from inflammatory leucocytes, not as a result of a specific cause or as a special

and exaggerated reaction of the constituent elements of nervous tissue, but rather as a result of the peculiar progress and mode of evolution of the inflammatory process. H. DOUGLAS SINGER.

**THE PRESENCE AND SIGNIFICANCE OF CHOLIN IN THE**

- (9) **CEREBRO-SPINAL FLUID IN EPILEPSY AND ORGANIC DISEASES OF THE NERVOUS SYSTEM.** DONATH, *Hoppe-Seyler's Zeitsch. f. Physiol. Chem.*, Bd. 29, Heft 6.

THE author adduces evidence in favour of the toxic theory of idiopathic epilepsy, and shows that in the great majority of cases (19 times out of 22) cholin is present in the cerebro-spinal fluid. His technique differs slightly from Mott's latest method (for the blood), but consists essentially in the formation of a cholin chloroplatinate, the crystals of which are readily recognisable under the microscope. Clinically the test is quite practical. The injection of chemically pure cholin into the brain of dogs produced convulsions. Under exactly similar conditions the injection of hydrochloric acid produced little or no effect. In simple hysteria, in hysterio-epilepsy, and in neurasthenia the cholin test was negative. The author concludes that cholin must play an important rôle in the production of epileptic convulsions. In several cases of dementia paralytica, tabo-paresis, and tabes, characterised by recurrent paralytic seizures, the cholin test was positive.

Similar results have been obtained recently by Mott.

S. A. KINNIER WILSON.

**THE MORPHOLOGY OF MUSCULAR DEGENERATION.** D. DE

- (10) BUCK and L. DE MOOR, *Névraxe*, 1903, Vol. v., p. 229.

THE majority of anatomists look upon the degeneration of muscle as a purely passive process, as regards the muscle fibres themselves, the active rôle being taken by the connective tissue elements of the perimysium. Many writers have noted a multiplication of the muscle nuclei, without, however, seeming to attach much importance to this. In this paper Buck and Moor describe the appearances met with in the successive stages of degeneration of muscles, whose nerve supply had been cut off in various ways. The animals experimented on were allowed to live from ten days to three months, and portions of the affected muscles were fixed either in 10 per cent. formalin or Marchi's or Fleming's solutions and stained by either von Gieson's or Heidenhain's methods. Ten days after the lesion the affected muscle showed, sometimes atrophy, sometimes hypertrophy of its fibres; in any case there was

always proliferation of the nuclei. The striation, both longitudinal and transverse, was less clear than normal. The nuclear division appeared to occur directly, no mitotic figures being seen in any specimens. Twenty days after the operation the muscle was generally atrophied, hypertrophy of the fibres being rarely seen now; the multiplication of the nuclei was more marked, and these lay in small clear spaces, having the appearance of vacuoles. Ricker and Ellenbek regard these vacuoles as due to œdema, but the present authors look on them as the result of a bio-chemical (digestive) action of the nuclei, and to this action they ascribe the chief part in the series of processes of muscle degeneration. At this period the nuclei were no longer confined to the region under the sarcolemma, they were also found in the centre of the fibre; it follows, then, that if these nuclei exert their digestive (sarcocytic) power, longitudinal cleavage of the muscle must occur. The peripheral nuclei act in the same way, and end by either digesting or breaking through the sarcolemma and passing out into the spaces between the fibres. In this way cleavage and exfoliation of the fibre occurs as the result of a sort of autophagocytosis of nuclear origin. By the sixth week all these changes were more marked, and the muscular substance was in many places represented by a mere meshwork, which still, however, showed faint striation; there was now a great tendency to revert to a cellular (embryonic) type of structure. Even after six months some relatively undamaged fibres could be found, although most had been converted into cellular units, some of these being still fibroblasts, while others were connective tissue cells, and others fat corpuscles. Buck and Moor conclude from these results that the old theory of muscular atrophy, which was based on a chronic inflammation of the connective tissue, causing sclerosis and ultimate disappearance of the muscle fibres, is quite incorrect; it is not the connective tissue but the muscle itself which reacts to the loss of functional or trophic stimulus. The divided nuclei surrounded by the remaining protoplasm constitute independent cells of the embryonic muscular type (*régression cellulaire*, Durante). These cells are true sarcoblasts, and if they receive proper (functional) stimulus can reproduce muscle fibres; in the absence of this stimulus they degenerate further, either into connective tissue or fat cells.

In the second part of this paper Buck and Moor describe the conditions of the muscles in various nervous diseases of cranial, spinal, or nerve origin. In adult and infantile hemiplegia and in pachymeningitis the appearances were identical with those arrived at experimentally; in the degeneration accompanying general paralysis a curious difference was noted: the sarcoplasm appears to undergo a fatty change without any definite cellular

arrangement into units occurring, and complete and final degeneration of the muscle soon supervenes. So also in a case of hydrocephalus of syphilitic origin, cellular formation was quite absent, and the nuclei showed hyperchromatosis and tended to run together into masses as in toxin poisoning (pynose, Schmaus and Albrecht).

As regards spinal cord injuries and diseases, cases of rupture of the cord, locomotor ataxy and poliomyelitis were examined; the results were all similar here, showing nuclear division and sarcolysis.

In a case of peripheral neuritis of rheumatic origin the nuclear multiplication and its effects in producing cleavage of the fibres were well seen; no trace of connective tissue reaction (sclerosis) was met with. In a case of Erb's scapulo-humeral type of muscular atrophy, fragments of the living muscles were removed and examined; the same proliferation of nuclei with sarcolysis and cleavage of fibres was found, while other fibres showed all degrees of atrophy or hypertrophy.

Finally, muscles suffering from the invasion of tumours (*e.g.* the pectoralis in a case of cancer of the breast), were examined and the same process was found at work, ultimately leading to the disappearance of muscular structure and connective tissue formation.

Buck and Moor conclude by pointing out that the metaplasia of the cellular units derived from the degenerating muscle has deceived most previous observers, leading them to ascribe the active part in the change to the connective tissue of the perimysium. Buck and Moor agree with Durande in regarding the return to the embryonic form of muscle structure as protective, and tending to preserve the power of reproduction of the tissue; for this reason they emphasise the importance of using a proper (functional) stimulus as a therapeutic agent in the treatment of muscular atrophies.

HEWAT FRASER.

**THE BRADSHAW LECTURE, ENTITLED "SOME OBSERVATIONS ON TUBERCULOSIS OF THE NERVOUS SYSTEM."**

(11) **TIONS ON TUBERCULOSIS OF THE NERVOUS SYSTEM."**

E. F. TREVELYAN, *Lancet*, Nov. 7, 1903, p. 1276.

BASING his remarks on the records of 114 fatal cases which have occurred during the past twenty years in the Leeds General Infirmary, Trevelyan deals with the subject of tuberculosis of the nervous system under these four headings:—

1. *The forms which the tuberculous infection may assume in the nervous system.*—These consist of tuberculosis of the dura mater, tuberculous meningitis in its more general and limited forms, tuberculous masses in the brain and cord, and a possible miliary tuberculosis of the brain itself. In 9 of his cases localised

thickening was noted over a limited area of the cortex, corresponding to the limited meningitis of French writers. Tuberculous masses were found in the brain in 33 of his cases: they were multiple in 17; in 23 of the 33 cases tuberculous meningitis was present—the tumours most often project on to the surface, hence infection of the cerebro spinal fluid is easily brought about. 2 cases of tuberculous tumour of the spinal cord, 2 cases of the relatively rare tuberculous tumours of the dura mater, and 2 cases with miliary tubercles of the dura mater were found. In connection with the question, lately much discussed, as to whether it is possible to have a tuberculous meningitis without the presence of tubercles, or at any rate with only a minimal tubercle formation, he finds that among the 114 fatal cases there are 14 in which thickening or exudation is noted at the base of the brain without visible tubercles—cerebral symptoms were present in 12 of these, and abundant tuberculous lesions elsewhere in the body in all. He therefore thinks it “fair to conclude that tubercle formation was at its minimum or even absent, at any rate in some of these cases, and that the changes noted were a manifestation of the tuberculous infection, and were responsible for the symptoms.”

2. *Mode of infection of the brain and meninges.*—Our knowledge is not at all complete. Almost invariably an older tuberculous focus will be found somewhere in the body—in only 6 of the 114 cases no such focus was found, and “here the notes are incomplete.” Direct infection through the nose and by extension from tuberculous disease of the ear is mentioned: obvious ear disease, not proved to be tuberculous, was present in 10 of the 114 cases: experimentally the writer believes that it is very difficult to infect the brain and meninges through the accessory cavities. A general tuberculous meningitis in spinal caries is of infrequent occurrence—only 4 such cases occurred in his series; he also mentions one very clear case of spread from cranium to the frontal region of the brain. The lungs were normal in 28 of his cases; miliary tuberculosis was found in 54, phthisis in 18 cases; he has had very little experience of tuberculous meningitis as a complication in obvious phthisis. Joints and bones were affected in 23 cases, the kidneys in 4, the generative organs in 2; tuberculous peritonitis was noted in 8 cases, intestinal ulceration mostly recent in 28. The records regarding the condition of the lymphatic glands in his series of cases are incomplete. One important point is referred to, viz.: that the glands may contain virulent tubercle bacilli and yet appear as healthy to the naked eye. Statistics are quoted to show that the mediastinal glands stand in the most direct relation to tuberculous meningitis—this can only be connected with the fact that the lungs constitute the port of entry of the tubercle bacillus in the large majority of these cases; infection of the

mesenteric glands in a retrograde fashion from the mediastinal glands is an anatomical possibility.

In 8 cases, tuberculous meningitis occurred in from one to six or seven weeks after operation (on glands, joints, etc.): while there is no satisfactory explanation, the view that the tubercle bacilli have been liberated in the field of operation and have then gained access into the blood current should receive practical attention. Cases of tuberculous meningitis following rapidly on injury are referred to.

As regards the route by which the tubercle bacilli travel from the infecting focus to the meninges or brain, Trevelyan thinks that the striking changes found in the blood-vessels (blocking of artery, tubercles in the vessel walls) in tubercular meningitis along with the experimental evidence—that the meninges may be infected from the carotid artery, but not through the lymphatic system—“make it appear almost certain that the tubercle bacilli arrive by way of the blood-stream.”

3. *Some points in diagnosis connected with spinal puncture.*—Notes are given of the results of examination of the cerebro-spinal fluid from 5 cases of tuberculous meningitis. In none of the cases was the writer able to find the tubercle bacilli in the deposit; in 4 cases lymphocytes only were found, and in 1 case only polymorpho-nuclear cells; the inoculation test gave a satisfactory result in 3 out of 4 cases, and is as yet incomplete in the fourth case. The finding of the tubercle bacillus in the deposit is by far the best test for clinical purposes: it has given positive results up to one-half to three-fourth of cases; his own failure is looked on as a matter of accident. The inoculation test is also very valuable but takes time, and, even if it should give a negative result, he looks upon the finding of an acid- and alcohol-fast bacillus resembling the tubercle bacillus in the deposit as conclusive of the nature of the meningitis. With regard to lymphocytosis in tubercular meningitis, so much written about lately, it is pointed out that lympho-cytosis has been found in cases of tuberculous tumours without meningitis; also, in one of the writer's own cases, only polymorpho-nuclear cells were found, but the inoculation test settled the diagnosis, thus confirming Leri's remark that, if the clinical indications and the cyto-diagnosis are not in agreement, the latter is not always right.

The study of the spinal fluid—aspiration being avoided—is advocated as a safe measure and a valuable means of differential diagnosis of the various forms of meningitis.

4. *Recovery from tuberculous affections of the nervous system.*—The writer refers to the few cases in literature which establish “the very slender possibility of recovery from a tuberculous tumour” and there are possibly two such in his own series: one

must, however, bear in mind the possible development of other masses in the brain, or the child may die of tuberculous meningitis or other form of tuberculosis.

As regards recovery from tubercular meningitis, although there are some remarkable cases on record—references to which are given, with the grounds, anatomical or clinical or bacteriological, on which the diagnosis of the nature of the meningitis was made—"the prognosis must still be looked upon for practical purposes as hopeless": spinal puncture, though possibly of value in a simple meningitis, is of quite doubtful utility in tuberculous meningitis. Prophylaxis deserves the closest attention: the danger which a careless phthisical patient is in his own house has been placed beyond doubt and points to the necessity of notification and isolation of consumptives.

A. W. MACKINTOSH.

## CLINICAL NEUROLOGY.

### PARALYSIS OF THE SPINAL ACCESSORY NERVE IN TABES

(12) DORSALIS. SEIFFER, *Berlin klin. Wochenschrift*, 1903, Nos. 40 and 41.

SEIFFER, in recording three cases of the above rare complication of tabes, discusses in the first instance the peripheral distribution of the spinal accessory nerve. He disagrees with the older view that the so-called "internal" branch of the spinal accessory supplies the larynx, palate and upper pharynx in whole or in part. He points out that "external" accessory palsy, involving the sterno-mastoid and trapezius muscles, is of extreme rarity, only nine cases, including his own three, being recorded, while cases of paralysis involving the laryngeal, palatal and pharyngeal muscles, are of very frequent occurrence, examination of available statistics showing that from 12 to 14 per cent. of tabetics suffer from laryngeal paralysis. He concludes, from a clinical point of view, that if the laryngeal muscles were innervated by the spinal accessory nerve, as well as the sterno-mastoid and trapezius, these latter muscles would be paralysed together with the laryngeal muscles much more frequently than they are.

His first case was one of tabes of four years' duration, complicated by severe degenerative paralysis of the right trapezius. The sterno-mastoid was not involved and no other complications were present. The case was regarded as one of partial accessory paralysis. The author discusses briefly the question as to whether the cervical nerves take any part in supplying the trapezius or not, and is of opinion that a definite decision is not possible, although he inclines

to the view that individual variations in the nerve supply of the muscle exist.

The second case was one of tabes of nine years' duration, complicated by paralysis of the right trapezius, sterno-mastoid, the right side of the soft palate, larynx and pharynx. Seiffer regarded it as one of affection of the right spinal accessory and vagus nerves.

The third case was also one of involvement of the spinal accessory and vagus in a woman.

From a review of the literature, Seiffer finds that the severity of accessory paralysis may vary greatly, sometimes amounting to slight involvement of one muscle, sometimes to complete paralysis of both muscles supplied by the nerve. With one exception, all the recorded cases were complicated by simultaneous affection of the vagus, an association easily explained by the proximity of the origin and course of the two nerves.

The author concludes by discussing whether the affection is a nuclear or peripheral lesion, and is of opinion that it is a peripheral nerve lesion.

J. W. STRUTHERS.

#### **CAN A SPINAL CORD TUMOUR DIMINISH IN SIZE SPONTANEOUSLY? S. E. HENSCHEN, *Mittel. aus den Grenzgebieten***

(13) *der Med. u. Chir.*, Bd. xi., H. 3, 1903.

THE patient was a man, aged 43, in whom in 1876-77 multiple subcutaneous tumours began to develop. Several of these were excised in 1882 and again in 1884-86, and found to be fibro-neuromata. In 1889-90 he first noticed stiffness and pain in the neck, and in 1890 stiffness in the right knee. Then sensory disturbances and later muscular wasting appeared in the right arm. In 1892 there was severe rigidity of the whole body with increased reflexes, and later a general paresis of the arms, trunk and legs with anæsthesia, bladder trouble and cilio-spinal changes. There was no evidence of vertebral disease.

A diagnosis of pressure paraplegia, due to a spinal tumour similar in character to the subcutaneous tumours above referred to, seemed probable. Cases of neuroma of the spinal cord have been described by Raymond, Schlesinger, Kahlen and Czerny.

The patient was opposed to operation, and even had he consented the case did not appear a favourable one, for it was quite possible that more than one tumour was present, and even supposing there was only one tumour which was removable, others might develop at a later date.

In November 1894 he began to improve, and continued to do so slowly. In May 1896, the anæsthesia had almost disappeared,



and he had regained considerable motor power, so that he was able to walk some distance.

The case is most elaborately reported and the diagnosis considered in very great detail.

EDWIN BRAMWELL.

#### **A CASE OF SYRINGOMYELIA WITH OHEIROMEGALIA.**

(14) A. SCHLITTENHELM, *Neurolog. Centralbl.*, Nov. 1, 1903, p. 1006

THIS case of syringomyelia is noteworthy owing to the presence of hypertrophy and trophic disturbances of the hands, and to the absence of muscle wasting.

A woman of 37 had for nearly twenty years been aware of a feeling of stiffness in the right hand, with loss of perception of pain and temperature, while for four years there was complete loss of all forms of sensation. The hand gradually increased in size from the time the affection was first noticed. The left hand had been becoming weak and analgesic for about two years, and had been getting larger for about six months. Purulent but painless ulcers frequently appeared on each hand from the time of their affection.

On examination, besides disassociated sensory loss and limitation of the power and range of movement of the hands and fingers, the hands were seen to be extremely large and marked with painless ulcers and scars of ulcers. There was great thickening of the skin and subcutaneous soft tissues covering them, and absence of the finger nails and distal phalanges.

When examined by the Röntgen rays the two distal phalanges of each finger were seen to be to a great extent disintegrated, while the basal ones were either of normal size or too small, so the enlargement of the hands is only to be ascribed to the thickening of their soft tissues, while in contrast the bones were either atrophic or disintegrated.

GORDON HOLMES.

#### **PARTIAL PARALYSIS OF ONE UPPER LIMB RESULTING**

(15) **FROM A VASCULAR LESION OF THE CORD.** W. G.

SPILLER and T. H. WEISENBURG, Univ. of Pennsylvania, *Med.*

*Bull.*, June 1903.

THE patient was a man aged sixty-six years, who at the time of his admission into hospital complained of having been unable to use the right upper limb for three days. Only an imperfect history was obtained, and unfortunately the man died before a thorough examination of his condition could be made; but the movement at

the shoulder-joint was very limited, whilst that at the elbow was fairly good. He was able to place his right forearm over the abdomen, to move the fingers fairly well, and with them to touch his face. The case was thought to be possibly due to a lesion of the brachial plexus.

Microscopical examination of the spinal cord showed that the change was chiefly localised to the eighth cervical and first dorsal segments on the right side; in this region there was slight sclerosis in that portion of the lateral column adjacent to the grey matter, and the lateral and medial portions of the anterior horn showed great reduction in the medullated fibres. Moreover, the nerve cells had entirely disappeared from the lateral region of the anterior horn. The vessels throughout this area were much sclerosed, and there was considerable proliferation of neuroglia, but no discoverable Marchi change.

The authors consider that the brachial palsy is explained by the destruction of the lateral cell-group of the anterior horn, and they refer to cases of progressive muscular atrophy and amyotrophic lateral sclerosis previously described by Stanley Barnes, and Mott and Tredgold, in which pronounced atrophy of the antero-lateral group was also present, the other cell-groups being practically unaffected. Bikeles and Franke also were able to demonstrate that in the dog, section of the peripheral nerves resulted in alteration in the dorso-lateral group of anterior horn cells. This case, therefore, corroborates the view that it is the lateral and dorsal groups of anterior horn cells which are chiefly concerned in motor function.

A. F. TREDGOLD.

#### **BUCCAL PERFORATION OR MULTIPLE NECROSIS OF THE**

(16) **MAXILLA IN A TABETIC.** J. CHOMPRET, *Arch. gén. de Med.*, Dec. 1, 1903, p. 3009.

THE trigeminal nerve may be affected in tabes, and more particularly the superior and inferior maxillary divisions, which contain sensory and trophic fibres. The buccal anæsthesia may therefore be accompanied by a progressive alveolar absorption and by a neuritic arthritis of the dental articulations, with weakening and atrophy of their ligaments.

The writer agrees with M. Galippe that a polymicrobial infection, buccal in origin, produces pyorrhœa alveolaris on this propitious soil, the ligaments are destroyed, the teeth fall out, and if the infection continues periostitis and necrosis of the maxilla follows. M. Chompret, however, believes that cicatrization may follow edentation, and subsequently traumatism by the teeth of the lower jaw may injure the mucosa, and either setting up a

traumatic neuritis or allowing a secondary infection to occur, produces ulceration, followed by periostitis and necrosis of the bone and the resulting perforations.

The clinical history preceding these conclusions details the case of a tabetic aged 45 with multiple buccal perforations. Painless and spontaneous loss of teeth was followed by necrosis of the maxilla, and perforations appeared in the incisor, palatine, and left molar regions, with nasal and maxillary sinus communications.

The teeth in the portion of the alveolus now occupied by the molar perforation were lost years before the tabetic symptoms appeared, and this site, together with that of the incisor ulceration, is placed opposite teeth that still remain in the lower jaw. The possibility of specific or gummatous antecedents is negated by the clinical history.

GOLDWIN HOWLAND.

#### **A CASE OF ASSOCIATION OF TABES WITH GENERAL PAR-**

**(17) ALYSIS OF THE INSANE.** JOFFROY and RABAND, *Rev. Neurolog.*, November 30, 1903, p. 1081.

THOUGH Joffroy has insisted that in the great majority of the cases of general paralysis which have symptoms of tabes the medullary lesions differ notably from those of ordinary tabes, typical tabetic changes are occasionally found in general paralysis. Such a case is here described.

A woman who had led an irregular life began to notice defective vision (from primary optic atrophy) and to suffer from lightning pains in the lower extremities in her twenty-eighth year. Later her gait became typically ataxic, a girdle sensation appeared, and there was some loss of sensation in the lower limbs. The pupils were found unequal and inactive to light and all deep reflexes were lost. The mental symptoms, which were of much shorter duration, were typical of general paralysis.

Changes characteristic of general paralysis were found in the cortex, and tabetic changes in the dorsal columns of the cord. In the lower lumbar region there was scarcely a normal fibre in the entering dorsal roots, and the dorsal columns were intensely degenerated, save in the commissural zone. In the middle of the lumbar enlargement the middle and lateral root zones were severely affected, but the commissural only slightly, while in the upper lumbar region the degeneration of the dorsal roots was not so complete, and a group of normal fibres was visible in the middle root zone bordering on the entering roots. Above this level the amount of degeneration in the dorsal columns corresponds to the state of the entering roots, besides depending on the dis-

tribution of the fibres ascending from the degenerated roots of a lower level. As the roots were only slightly affected in the lower half of the dorsal cord and were relatively intact above it, besides the severe sclerosis of Goll's columns there was only some degeneration in the middle radicular zone, and these two areas of degeneration gradually approach each other towards the cervical cord. The intramedullary change is thus a regular system degeneration dependent on the changes of the dorsal roots as in tabes, and is contrasted by the authors with the more diffuse focal degeneration in which the endogenous tracts are not spared, characteristic of general paralysis, and they regard the case they publish as one of true tabes associated with general paralysis.

GORDON HOLMES.

**A CONTRIBUTION TO THE STUDY OF THE VOICE IN THE  
(18) FIRST AND SECOND STAGES OF GENERAL PARALYSIS.**

E. MARANDON DE MONTYEL, *Journ. Neurol.*, Nov 5, 1903,  
p. 495.

IN some general paralytics the initial symptom has been the loss of a beautiful voice, and where the disease occurs in singers the voice often becomes nasal or guttural, modified in pitch, quivering, bawling, or quite hoarse. These changes have been referred to loss of power in the muscles of the vocal cords, palate, and larynx. (Schule.)

According to de Kraft-Ebing the voice muscles are often early affected by the paralysis and ataxy; the voice becoming hoarse, hollow, and muffled; and, if the innervation of the roof of the palate is affected, the voice assumes a snuffing character.

Mickle says the voice is perhaps hollow, rough and deep, and that the voice becomes feeble and monotonous when the tension of the cords is enfeebled and the expiratory force diminished; if, on the other hand, the tension of the cords is irregular, the voice becomes low in character. An elevated intonation is observed where the inflection of the voice is lost.

The author kept records during several years of observations on 55 men suffering from general paralysis, and limited his observations to the first and second stages of the disease on account of the difficulty in ascertaining the exact character of the phonation in the terminal stage.

The author's records consisted of 1596 separate observations, from which he drew the following conclusions:—

1. The voice in general paralysis is as often abnormal as normal.

2. Two alterations occur: the voice becomes *flat* or *sharp*; in the former it is hollow and muffled, in the latter it is like a woman's voice—or rather like that of a eunuch.

3. Alteration to *flat* is very common and presents three degrees: slight, moderate, and excessive. Of these the moderate is the most frequent.

4. Alteration to *sharp* is less frequent, and is always of excessive intensity.

5. The voice is more often changed in the first stage than in the second, though very nearly the same proportion of alteration to *flat* and *sharp* occurs in both stages.

6. The abnormal voice is most frequently observed in paralytics who are passing through the stage of remission, and the deviation in the voice is generally towards *sharp*. If *flat*, it is generally slight in degree.

7. Of the varieties of general paralysis, it is in the melancholic type that the voice is most commonly changed, and the alteration is usually towards *sharp*. If *flat*, it is generally excessive in degree.

8. In the other varieties the alteration is generally to *flat*. In the demented type the voice is more often normal than abnormal.

9. In cases where there is a history of syphilis the voice may be *sharp* or *flat*. If *flat*, the mild syphilitics generally show alteration to excessive *flat* and the acute syphilitic cases to slight *flat*.

10. Alteration to *sharp* or *flat* may occur with an alcoholic history. If *flat*, it is always moderate in degree.

11. Age influences the vocal troubles; they are less common in the young paralytics.

12. In the incipient stage of general paralysis the alteration in the voice is generally towards *sharp* or slight *flat*.

13. There is no connection between the motor disturbances and the voice troubles.

14. When the genital sense is normal or merely enfeebled, the phonation is usually normal.

15. When there is sexual impotence the voice is often *sharp*, or *flat* (excessive degree).

16. The alterations in the voice of the general paralytic are due to changes in the central nervous system, and are accentuated by the "cries" uttered in most cases of the disease.

The author also studied the evolution of the phonatory troubles in 37 paralytics during the first and second stages of their disease, and came to the following conclusions:—

1. The voice may be normal throughout, or it may become abnormal and remain so, or it may alternate between normal and abnormal.

2. Where the voice alternates between normal and abnormal, the normal periods are of indefinite length, and the abnormal periods are of longer duration than the normal.

3. The tendency of the voice to alter itself is neither augmented nor diminished by the progress of the disease; but the vocal troubles are not so fixed in the first stage as in the second, nor is their intensity so marked.

4. The evolution of the different degrees of intensity of the vocal troubles is as variable and as capricious as the evolution of the troubles themselves.

In one of the author's cases the voice was at first *sharp*, then became *flat* after an intervening normal period. The two-toned voice was never observed.

H. DE M. ALEXANDER.

**THE DIAGNOSTIC SIGNS OF TUMOURS OF THE OCCIPITAL  
(19) LOBE AND OF THE TEMPORO-SPHENOIDAL LOBE. H.  
DURET, *Rev. Neurolog.*, Nov. 15, 1903.**

*Tumours of the Occipital Lobe.*—The characteristic sign of a lesion in this situation is homonymous lateral hemianopsia, which may exist without other localising signs, but which is, in the majority of cases, associated with other signs—aphasic troubles, word blindness, hemianæsthesia and hemiplegia.

Where uncomplicated hemianopsia exists the localisation is certain, for this syndrome occurs only in lesions of the calcarine cortex, *i.e.* the cuneus, the lingual, and the fusiform lobules.

Tumours extending deeply from the internal surface of the occipital lobe are apt to give rise to hemianæsthesia and hemiplegia. Growths involving the external surface, the white centre or the sub-cortical regions of this lobe, are very commonly associated with some variety of sensory aphasia, since the angular gyrus may be involved, and the connections of the occipital lobe and angular gyrus with the auditory word centre and with the frontal lobes may be destroyed.

(The author does not mention visual hallucinations, visual word hallucinations nor epileptiform attacks with a visual aura, as symptoms of disease of the occipital lobe.)

*Tumours of the Temporo-sphenoidal Lobe.*—These are grouped into three classes, according to the symptoms resulting:—

1. The chief physical signs are deafness and word deafness.

The author considers deafness of the opposite ear an important sign of a temporo-sphenoidal lesion. Deafness to certain tones only may exist. Auditory hallucinations and epileptiform attacks with an auditory aura are most important signs and are of certain

localising value. He suggests that epileptiform attacks having as a constant aura a sharp deviation of head and eyes to one side may be an important sign of temporo-sphenoidal disease.

2. The chief symptom is sensory aphasia. Attention is drawn to the interesting nature of early psychic peculiarities; for example, the author recites the case of an architect who lost all cognisance of the nature of geometrical drawings.

3. Tumours which reveal their situation by the implication of the crus cerebri and the adjacent nerves.

The situation is usually the internal aspect of the temporal lobe. Loss of taste and smell may occur, and olfactory and gustatory hallucinations may be present, either alone or as the warnings of epileptiform attacks.

The author suggests that the hypotheses of Touche, that the anterior parts of the fusiform and lingual lobules may be centres concerned with topographic memory and with the faculty of orientation, may in the near future receive such confirmation as to place other valuable localising signs at the disposal of the clinician.

JAMES COLLIER.

# **THE PROJECTION OF THE RETINA ON THE CALCARINE (20) CORTEX. S. E. HENSCHEN, *Semaine Med.*, April 22, 1903.**

THE localisation of the visual cortical centre is a question towards the solution of which Professor Henschén has made valuable contributions. In this paper he expresses the opinion that deductions which are based exclusively on the method of degeneration must be inconclusive, since it does not follow that all the degenerated fibres are directly concerned with vision. Experiments on animals are of little value, since no animals possess a sufficient degree of intelligence to allow of delicate observations regarding localised alterations of the fields of vision. Hence it follows that further information as to retinal cortical representation is only to be expected from clinical and anatomical observations upon cases of localised lesions in the human subject.

The author refers to his well-known case in which a lesion limited to the calcarine cortex produced an almost complete homonymous hemianopia. Lesions limited to the cortex cerebri other than the immediate neighbourhood of the calcarine fissure do not produce hemianopia. The author then mentions the salient points in some other cases which have come under his observation. In one case, a localised lesion which involved almost the whole calcarine cortex and subcortex, and was limited to these parts, was associated with homonymous hemianopia. Another case is referred

to in which there was destruction of the dorsal portion of the lateral geniculate body, with a degeneration of the upper part of the optic radiations. In this case there was a hemianopia affecting only the corresponding inferior quadrant of the visual field. A third case resembled very closely that just described. In this case the geniculate ganglion was destroyed, and there was a secondary degeneration of the optic radiations, only the inferior fibres escaping. The unaffected fibres could be traced from the inferior part of the ganglion to the lower lip of the calcarine fissure. The hemianopia was of the quadrant type, involving the inferior quadrant of the field.

These facts demonstrate that definite areas of the retina are represented in definite cortical areas, the upper lip of the calcarine fissure corresponding to the upper part, the lower lip to the lower part, of the retina.

Another most interesting case is described in which, in addition to a superior quadrant hemianopia, there was an *absolute* homonymous scotoma limited to a portion of the lower quadrant of the same side of each field. At the autopsy a softening of the inferior calcarine lip was found, and in addition another area of softening of the superior lip.

A further case is mentioned in which a lesion, situated at the bottom of the calcarine fissure, was associated with a homonymous scotoma with its long axis in the horizontal meridian.

The paper, which is illustrated by numerous charts of the visual fields, and drawings showing the position of the lesions in the various cases referred to, is an important contribution to the subject.

EDWIN BRAMWELL.

**ON THE DIAGNOSTIC VALUE OF THE "LIDSCHLUSS-  
(21) REACTION" OF THE PUPILS.** A. WESTPHAL, *Neurolog.  
Centralbl.*, Nov. 15, 1903, p. 1042.

In a man of 53, who developed complete paralysis of all functions of the one oculo-motor nerve after an injury to the head, though the light-reflex of the pupil was lost, it contracted on strong action of the orbicularis palpebrarum. This phenomenon persisted during the recovery of the functions of the oculo-motor nerve, and was finally the single abnormal sign present.

The author concludes that defect or loss of the light reaction of a pupil is the most favourable condition for the appearance of this phenomenon, to which attention has chiefly been directed by himself and Piltz, and would regard it as a delicate test of unilateral



defective light-reflex as is frequently present in general paralysis of the insane.  
GORDON HOLMES.

**A CASE OF RECURRENT PARALYSIS OF THE THIRD NERVE.**

(22) WILLIAM GEORGE SYM, *Ophth. Rev.*, Nov. 1903, p. 303.

THE author describes a case of third and sixth nerve paralysis occurring with successive pregnancies. During the patient's first three pregnancies the eyes gave her no trouble. A month before her fourth child was born the sight, she says, was dim, and for a fortnight she saw double. The symptoms disappeared a week after her confinement. For six weeks before her fifth confinement there was double vision and partial left-sided ptosis. Complete recovery had taken place by the time the child was a week old. Nine or ten weeks before the close of the sixth pregnancy, diplopia again developed with ptosis on the left side. Dr Sym, who saw the patient before the birth of this child, found almost complete paralysis of the left sixth nerve with very marked affection of the branches of the third which supply both the external ocular muscles and pupil. The movements of the right eye were unaltered. The fundus of each eye was normal. The patient had suffered from headache, which was felt all over the head, and not hemicranial in type. She stated that her left eye felt numb, but the author was unable to satisfy himself that there was any distinct objective sensory defect.

It is now two years since the patient's sixth child was born, and although great improvement has taken place, the paralysis has not yet completely disappeared.  
EDWIN BRAMWELL.

**TWO CASES OF TUMOUR ON THE AUDITORY NERVE.**

(23) J. LEFINE, *Rev. Neurolog.*, Nov. 30, 1903, p. 1104.

THE two cases described are practically identical. In each a tumour somewhat larger than a pigeon's egg, and histologically of fibro-sarcomatous nature, was attached to the auditory nerve at its entrance into the internal auditory meatus.

The symptoms were progressive unilateral deafness and tinnitus in the same ear, facial paralysis, headache, acute optic neuritis, vertigo (character not described), and a reeling gait, with a tendency to fall to the same side.

In differential diagnosis from cerebellar tumours, the early complete deafness and the existence of facial paralysis must be relied on.

GORDON HOLMES.

# **FAICIAL SPASM: ITS DISTINCTIVE CLINICAL CHAARACTERS.**

(24) HENRY MEIGE, *Rev. Neurol.*, October 31, 1903.

UNDER this name the author describes attacks of painless spasm of one side of the face which are quite distinct from Tic. He divides the clinical characters into three degrees or stages of development: (1) There is a sudden onset of quivering at the free border of the lower eyelid which spreads fibril by fibril to the whole lower orbicularis and thence to the upper lid; as the spasm increases the two lids become drawn together, but the palpebral fissure is rarely completely occluded. After a varying period, the contractions gradually cease in an inverse order. (2) Beginning in the same manner the attacks are more frequent and more severe, and the spasm rapidly spreads to the other muscles of the same side of the face, affecting a few fibrils of one, a fascicle of another, and the whole of a third, until the whole side of the face is thrown into a condition of trembling tetanus to which the author gives the name "*contracture frémissante*." After a varying period the spasm gradually passes off. Sometimes the pillars of the fauces and the uvula may be seen to partake in the spasm and there may be also a few transient and wandering fibrillary contractions on the opposite side of the face. Occasionally there is also some vasomotor disturbance on the affected side of the face. (3) Finally the periods of intermission may become inappreciable, so that one side of the face presents the "quivering contracture" as a permanent condition.

The distinctive features of the condition are: (1) The entire absence of pain. (2) The grimace produced does not mimic any form of emotional expression. (3) The sudden onset, gradual spread, varying period of apogee, and gradual relaxation. (4) Attacks may be continued into sleep, and may arise during sleep. (5) Voluntary effort has no influence in checking the attacks when once started. (6) Psychomotor exercises so valuable in the treatment of habit spasms are here of no avail.

Having given an excellent description of nine cases (three of which have been previously published by himself and one by Marie), the author discusses three cases of Facial Spasm associated with Myokimia described by Bernhardt, Newmark, and Frenkel, which he is inclined to think belong to the same category, and in conclusion points out that facial spasm must not be hastily deemed hysterical, even when occurring in a hysterical patient.

H. DOUGLAS SINGER.

**PROGRESSIVE FACIAL HEMIATROPHY.** FISCHER, *Monatsschr.*  
(25) *f. Psychiat. u. Neurol.*, 1903, p. 366.

ON the case which forms the subject of this communication, Fischer bases a new theory of the pathology of this obscure condition. The patient was a boy aged twelve, of healthy ancestry. He had suffered from scarlet fever at the age of five; no history of trauma. When he was six years old a brownish spot, of gradually increasing size, was noticed on the right side of the neck; when he was about nine his right ear seemed smaller than his left; a year later a spot appeared at the side of the nose, and a notch on the upper lip a little later. Apart from the facial hemiatrophy, no abnormalities worthy of note were present. The skull was symmetrical. The two halves of the face were markedly unequal, the right half, from the zygoma down, being the smaller, this being solely due to atrophy of the soft parts, the facial bones being apparently of equal size. The right naso-labial fold was the deeper, the nose was inclined slightly to the right, the right half of the upper lip was thinned and showed a depression (like slight hare lip) about a third of an inch from the middle line. The innervation and movements of the facial muscles were normal on both sides, except that after any movement the muscles on the right half of the face seemed to return to the position of rest somewhat slowly. Atrophy of the skin was present in two areas: at the angle of union of the right ala nasi with the cheek, and on the right side of the neck; the latter patch being much the larger, and extending up along the sterno-mastoid behind the ear. The atrophic skin had the usual appearance. The right ear was the smaller, and the right meatus the narrower of the two. The case therefore shows: (1) diminution on one side of the face, without alteration of the bones; and (2) the usual atrophic areas of skin, which had the interesting peculiarity of being situated at the sites of closure of the foetal clefts. In many of the recorded cases atrophic areas of skin have been described as occurring under the eye-lids, in the zygomatic region, in front of the ears, at the side of the nose, on the side of the neck—all situations where closure foetal clefts take place. Out of 77 cases Fischer found this to be the case in 20, in 13 the atrophic patches existed both at these sites and elsewhere, in 7 they were found only in positions where no foetal clefts occur, and in 37 their situation is not stated. Among the theories which have been propounded to explain the cause of facial hemiatrophy, that of its nervous origin, supported by the frequency with which other nervous symptoms accompany it, is rejected by Fischer, on the ground of insufficient evidence, and particularly because, as Moebius has pointed out, the very cases in which other nervous symptoms (paralysis of the fifth,

sympathetic, etc.) have been most marked, are those which lack the essential feature of true hemiatrophy—viz., the atrophy of the skin. Another theory is that the disease is an aplasia of the skin and subcutaneous tissues; another, that it is due to a toxin acting directly on the skin; and yet another, that trauma may play a part in its production. Fischer believes that, just as slight abnormalities in development may lead to pathological overgrowth at positions where foetal clefts unite, so similarly they may lead to retrogressive disturbances of nutrition. Since, however, in some cases the cutaneous atrophy is not limited to those sites, this theory by itself is inadequate. He therefore supposes that some toxin, either exogenous or endogenous, is at work in these cases, and that its activity is conditioned by: (1) developmental anomalies in the closure of the foetal clefts, or (2) injuries (e.g. traumata, neuritis) in later life which render certain areas of skin susceptible.

J. S. FOWLER.

**PROGRESSIVE FACIAL HEMIHYPERTROPHY.** HOFFMANN,  
(26) *Deutsche Ztschr. f. Nervenheilk.*, Bd. xxiv., H. 5 u. 6, 1903,  
p. 425.

As compared both with congenital hemihypertrophy and progressive facial hemiatrophy, the condition described by Hoffmann in this paper is very rare: he has only been able to find records of five cases in addition to his own. His patient was a girl of fourteen, whose family history was good, and who had suffered from no other disease. At the age of two years, just as dentition was complete, swelling of the right half of the face was noticed, but this appears to have varied from time to time, and occasionally the face resumed its normal aspect. The hypertrophy has only become really evident within the past two or three years, and for about the same time hair has grown freely on the right half of the upper lip. The essentials of the case are briefly as follows:—There is a marked enlargement of the right half of the nose, of the right cheek from the lower eyelid to the angle of the mouth, and of the mucous membrane covering the right half of the hard palate. The hypertrophy is sharply defined by the middle line; it is most marked in the right upper lip, which hangs down over the lower, and is also covered by a thick growth of hair. The bones are in no way affected; there is no alteration in the colour of the skin, nor any sign of vascular lesion. The forehead, tongue, soft palate and chin are symmetrical. There is no affection of the facial muscles, or of the muscles of mastication; common sensibility and the sense of taste are normal, as also are the other organs and functions. New growth, angioma, and congenital hemihypertrophy were easily excluded in making a diag-

**nosis.** Hoffmann gives a brief summary of the five other cases, and draws the following deductions from them: of six cases, four occurred in females, two in males; the right side was affected in four, the left in one, and in one case the side is not stated. In three cases the condition was preceded by a disease or injury (abscess of cheek, trigeminal neuralgia, wound of cheek), in one it occurred along with acromegaly, and in two no cause could be assigned. In the last it was noteworthy that the hypertrophy was first noted at the close of dentition. The condition seems due to a nervous lesion; in Hoffmann's own case it was limited to the area of the second division of the fifth; and the absence of vascular changes, affection of the growth of hair, and strict delimitation by the middle line, favour the view that it is due to trophic influence.

J. S. FOWLER.

**THE ETIOLOGY AND SYMPTOMATOLOGY OF INTERMITTENT**

(27) **CLAUDATION.** S. GOLDFLAM, *Neurolog. Centralbl.*, Nov. 1, 1903, p. 994.

**INTERMITTENT** claudation, an affection due to constriction or obliteration of the arteries of the lower extremities, to disturbed vasomotor function, and to relative ischæmia during muscular contraction, could not hitherto be ascribed to a definite cause.

The author, from his relatively large experience, has ceased to regard overwork as a factor, though this has been again recently proposed by Saenger, and he has not been able to find support for Erb's theory that it is due to excessive use of tobacco. He is now inclined to ascribe it to a nervous diathesis and hereditary disposition, and supports this view by reference to its occurrence in three pairs of brothers, to the fact that a large proportion of the cases have been described in Jews, and to its frequent appearance in young individuals with other evidence of vascular abnormality. Excess in tea and coffee too may have a rôle in its production.

The new symptom described is an abnormally easy exhaustion of the muscles resembling that in myasthenia gravis, but to be distinguished from that of the latter affection by the simultaneous acceleration in the rate of the pulse and respiration, and by the contrast between the firmly contracting muscles and the slight motor effect.

GORDON HOLMES.

**THE ETIOLOGY OF INTERMITTENT CLAUDATION.** IDELSOHN,

(28) *Deutsche Zeitsch. f. Nervenheilk.*, Bd. 24, S. 285.

**THE** author gives in considerable detail the symptomatology of fourteen cases occurring in his general practice, diagnosed as cases

of intermittent claudication. He is careful to point out that the diagnosis is essentially a clinical, and not an anatomo-pathological one. The condition is characterised by local syncope, recurring pains, very often absence of pulsation in the posterior tibial artery, and great chronicity. Several of his cases ended in gangrene. Of the fourteen, ten had single or double flat foot, and twelve were Jews. The author argues for a causal relation between flat foot and disturbances of the circulation, and believes that the underlying pathological factor in intermittent claudication is a constitutional weakness of the vascular system. An interesting if somewhat slender argument is advanced on the fact that so many of his cases occurred in members of the Jewish community.

S. A. KINNIER WILSON.

**THE DIAGNOSIS OF HYSTERIA.** PURVES STEWART, *Practitioner*, (29) Oct. and Nov. 1903.

THESE two lectures deal with some of the diagnostic features of hysteria. At the outset the reality of the disease is insisted on, and it is sharply marked off from malingering. Hysteria is regarded as a disease of nerve-metabolism, affecting not only the highest or psychical centres, but implicating the whole nervous system in varying degrees and producing symptoms, psychical, sensori-motor, visceral, vaso-motor, etc., corresponding with the parts of the nervous system affected. The basis of hysteria, as of all diseases, bodily or mental, is physical in nature. Not infrequently gross organic disease produces symptoms indistinguishable from hysteria. Hysteria, therefore, may co-exist with organic disease. But in practice the term is usually restricted to cases where no organic disease is present.

Amongst the psychical symptoms, which are to a greater or lesser degree invariably present, the most outstanding is deficiency of inhibition, so that the patient reacts too readily to stimuli or suggestions, whether arising in the outside world or within her own body. This explains many of the symptoms, notably the emotional tendency, the want of self-reliance and craving for sympathy. Hence the importance in treatment of removing the patient from her old surroundings to break the vicious circle. The various affections of speech and of articulation are referred to, including *diarrhœa verborum*, aphonia, stammering, etc.

Sensory symptoms are of greater diagnostic importance than is commonly realised. Amongst them are the various painful or hyperæsthetic areas, of which the commonest is that in the left inguinal region. The term "ovarian" applied to such a painful area is a misnomer, inasmuch as this symptom is equally frequent

in male and in female hysterics. Some painful spots may be hysterogenetic, pressure on them inducing a hysterical fit, others hysterofrematic, pressure on which arrests a hysterical fit if present. Hyperæsthesia may also affect the special senses, especially vision.

A degree of anæsthesia exists, if looked for, in the overwhelming majority of cases. Thus out of 62 cases, anæsthesia was present in 49, absent in 12, and the one remaining case showed unilateral hyperæsthesia. Such hysterical anæsthesia is often unnoticed by the patient. The cases where it is noticed spontaneously are chiefly those in which motor paralysis is also present, calling the patient's attention to the affected area. Charts are given, exemplifying the chief varieties in distribution of hysterical anæsthesia. The two main types are the hemianæsthetic and the segmental. Often the two types are combined. The well-known "crossed amblyopia" is described and its frequent association with loss of other special senses on the affected side. Monocular diplopia and polyopia are always hysterical. Micropsia and macropsia are also not uncommon.

The motor phenomena are classified into irritative and paralytic. Amongst the former, the various types of hysterical fits are described; reference is also made to hysterical trance, to somnambulism and to double consciousness. The different varieties of tics and habit spasms are illustrated. The main features of hysterical paralysis, monoplegic, hemiplegic or paraplegic, are also referred to. Glosso-labial hemispasm, though not common, is of great diagnostic value when present. The presence of muscular atrophy, even to an advanced degree, does not, of itself, negative the diagnosis of hysteria. Contractures also, of various types, may occur. True ankle-clonus does not occur in hysteria, though a pseudo-clonus is occasionally met with. The extensor type of plantar reflex is also regarded as one of the strongest evidences of organic disease.

The visceral and vasomotor phenomena of hysteria are exemplified. Amongst these are mentioned pseudo-angina, ærophagy or gulping of air, spasmodic stricture of the œsophagus, hysterical vomiting, rhythmic gastric movements, borborygmi, pseudo-pregnancy, phantom abdominal tumours, pseudo-appendicitis, hysterical diarrhœa, etc. The existence of pseudo-hæmoptysis and pseudo-hæmatemesis cannot be denied, but care should always be taken to exclude organic disease and malingering. Amongst the vasomotor phenomena, the great frequency of dermographism amongst hysterics is indicated.

Finally it is pointed out that hysteria and organic disease may co-exist in the same patient.

AUTHOR'S ABSTRACT.

**ON CERTAIN REFLEX MOVEMENTS HITHERTO LITTLE  
(30) OBSERVED WHICH OCCUR IN CASES OF INFANTILE  
SPASTIC DIPLEGIA. H. OPPENHEIM, *Monatsschr. f. Psychiat.*  
*u. Neurol.*, Bd. 14, H. 4, S. 241.**

THE writer calls attention to two reflex phenomena which he has found in cases of that form of spastic diplegia which he has himself described as infantile pseudo-bulbar paralysis.

The first of these consists in a spasmodic contraction of the muscles of the trunk and limbs which follows a sudden noise, such as may be made with a hammer. It seems like a great exaggeration of the usual start which normally results from such a cause, but it differs from this in several important ways. The reaction which accompanies a sudden start in a healthy person ceases to occur, or occurs only slightly, if the person is warned beforehand, or if the experiment is frequently repeated. In these cases, however, the spasmodic muscular contraction takes place to exactly the same degree, however often it is repeated, and it is quite as marked when the child is allowed to watch the hammer. The reflex may be elicited by suddenly grasping and shaking the skin of the abdominal wall, as well as by making a loud noise. A flash of light does not cause it. Although the contraction produced is violent, the child laughs and seems to like it.

The writer is of opinion that these facts are in favour of the reflex being regarded as due rather to an increase of the motor than to an exaggeration of the psychical reaction. He looks on the phenomenon as essentially due to an exaggeration of lower reflexes. He would explain its occurrence in spastic diplegia by the hypothesis that when large areas of the cerebral cortex are cut off by disease, stimuli acting through the sense organs tell with far greater force on the subcortical, bulbar and other lower centres.

The second reflex described is as follows:—When the lips or tongue or some part of the mouth or throat of one of these children is touched momentarily with a glass rod, a series of reflex acts take place. These consist of chewing, sucking and swallowing movements, during which the tongue is moved about in the mouth and then protruded between the teeth. The movements are repeated rhythmically at very short intervals, occurring as often as 25 to 30 times in the 10 to 20 seconds during which the whole reflex lasts.

The phenomenon represents an enormous increase, in both degree and extent, of the normal sucking reflex of early infancy prolonged into later childhood. It is, of course, quite morbid, and can effect no useful purpose. In spite of its apparent dissimilarity, its causation is probably to be explained in a very similar way to that of the other reflex described above.

JOHN THOMSON.



**TRICEPS, BICEPS, AND FINGER CLONUS.** T. H. WEISENBURG,  
(31) *Journ. of Nerv. and Ment. Dis.*, Nov. 1903.

AFTER referring to the fact that most of the text-books of neurology make no reference to these phenomena, the author describes three cases in which the above signs were present in spastic arm states.

STANLEY BARNES.

**THE PROBLEM OF THE WELL-TO-DO INEBRIATE.** CHARLES  
(32) L. DANA. Reprinted from *New York Medical Journal* for August 1 and August 8, 1903.

IN this paper Dr Dana first discusses what he considers the inadequacy of present methods of treatment of the well-to-do inebriate. The chief plans possible under present existing law in America are mismanaged. For the cure of the habit two things are considered essential: (1) That the patient be kept absolutely from opportunities of indulgence for from one to three years; (2) that during this period the patient be under restraint, and his time employed in improving by every possible means his bodily, mental, volitional and initiative power.

The drink crave in many cases presents a periodically explosive character, resembling in this other chronic nervous disorders such as epilepsy. One would not feel assured that an epileptic was cured after the cessation of fits for one year only. More confidence would be felt at the end of two years; while after three years' probation in the great majority of cases the patient could be regarded as cured. So also in many cases of alcoholism.

The author desires to see legislation by which (1) an inebriate could be committed for from one to three years, and (2) the establishment of colonies for the isolation and treatment of such cases. He acknowledges the special difficulties in the way of attaining the second of these conditions, and insists on the importance of having such isolated colonies made as attractive as possible, with every facility for work, study and amusement.

A. HILL BUCHAN.

## PSYCHIATRY.

**OBSESSIONS AND IMPULSIONS.** SERGE SOUKHANOFF, *Presse*  
(33) *Méd.*, Sept. 21, 1903.

IN this lecture, delivered before the University of Moscow, the author draws a sharp line between simple obsessions and impulsive actions.

Dealing first with the uncomplicated *ideo-obsessive* constitution, he states that these obsessions are seen in cases which exhibit a peculiar congenital *neuro-psychic* organisation, and may manifest themselves in various degrees, from a simple restlessness and excessive carefulness, to the most severe forms—the psychosis of obsessive ideas. But with this there are no signs of moral obliquity, and sexual perversions are very rarely met with.

The individuals suffering from morbid obsessions and phobias may be egoists, and may worry those around them, but they have a fairly just appreciation of their conduct, and their consciousness of their infirmity may cause them great suffering and remorse.

These obsessive ideas produce corresponding actions—the patient who suffers from the fear of infection or of filth will constantly wish to wash his hands, and he is disturbed if prevented from doing so, or he may make some special movement or gesture before he carries out simple actions. But so far the obsession contains nothing contrary to the patient's usual moral nature.

Sometimes, however, in what are called “obsessions of contrast,” such as the intrusion of erotic or obscene thoughts into his mind when he is engaged in some religious service, it might be thought that there was a moral as well as a mental twist; but as a matter of fact, there is always remorse at his inability to exclude these thoughts, which are purely *ideo-obsessive*, uncomplicated by any other pathological psychic condition.

In these cases the actions are not accompanied by any dangerous actions, but these last become possible when there is an accessory pathological state in which the voluntary inhibitions and moral sense are enfeebled. They are especially seen in senile dementias, in some functional cases—melancholias, and in cases of arterio-sclerosis of the brain.

To them he applies the term “impulsions,” and gives to it a signification entirely different to that associated with the term obsession; in fact the one excludes the other.

These impulsions are especially the result of psychic degeneration and psychic disequilibrium, but the patients do not exhibit the moral sensibility which is so prominent in the simple *ideo-obsessive* constitution, and it is a question whether impulsions are not always accompanied by some moral insanity. These impulsions are seen most frequently in chronic alcoholism, general paralysis, senile dementia and dementia præcox, and following epileptic attacks. They may also be isolated phenomena in the course of some manias and melancholias.

R. G. Rows.

**A FORM OF ACUTE HALLUCINATORY INSANITY IN  
(34) PRISONERS WITH DELUSIONS OF PERSECUTION,  
NEITHER FURTHER SYSTEMATISED NOR CORRECTED.  
E. RÜDIN, *Allg. Ztschr. f. Psychiat.*, vol. 60, 1903, p. 852.**

EVERYONE who has had to deal with criminal lunatics has felt difficulty in placing certain cases of insanity, arising in the prison, under any head in the usual classification. Rüdin has undertaken the praiseworthy task of submitting these cases to a more accurate examination. The following are their common prominent features: sudden onset in persons hitherto healthy-minded, in their second or third year of cellular imprisonment; aural, visual, rarely tactile hallucinations, with delusions of persecution on the part of the prison staff, not of a properly feeble-minded character, and with little systematisation. The bodily symptoms are headache, sleeplessness, over-excitability, loss of appetite, resulting in a feeling of sickness. The temper is peevish, suspicious, apprehensive, even furious or desperate, sometimes improving, but never becoming joyful. Orientation, interests and apprehension are good. Conduct is natural, writings correct. When the acute symptoms disappear after a few months or even a year, the system of delusions remains without being further developed; on the other hand the patient has no complete recognition of his disease.

The author points out the differential diagnostic criteria from paranoia, dementia præcox, the acute hallucinosis of Wernicke and of Kirm, epileptoid states and imbecility. The life of imprisonment is assumed to be the causal factor; by isolation the prisoner is excluded from diverting stimuli; therefore he will easily develop hallucinatory states. The prognosis is favourable; in the course of time, and soonest in liberty, the patient will recognise that he has been insane, and lose his irritability, but his power of psychical resistance will always be weak. It is not determined if these forms of insanity ever occur outside prisons.

[No more than a couple of years having elapsed since the onset of the disease in the reported cases, it may possibly be found by further investigations that at least some of such cases belong to dementia præcox and correspond to cases of heboïd (Kahlbaum), also called *formes frustes*, which not infrequently are met with outside prisons. The isolation may give the clinical picture its special colour; the same picture is, however, to be seen in jails in which no isolation is carried out. The deleterious influence of prison life upon the state of health probably arises more from troubles of digestion and the habitual anæmia which generally precede the first mental symptoms, than from isolation,

and from a consideration of all the clinical facts one is led to think that the insanity is probably due to a toxæmia depending upon a diminution of the resisting powers.—*Rev.*]

HANS EVENSEN.

## TREATMENT.

### A METHOD FOR THE RELIEF OF PAIN IN TUMOURS OF THE

(35) **BRAIN.** WILLIAM BROWNING, *Journ. of Nerv. and Ment. Dis.*, Nov. 1903.

IN this paper the author suggests the more frequent use of "vascular depressants, such as aconitia, veratrum, or gelsemium, in doses sufficient to soften and control the pulse." He claims that this method accomplishes quite as much as symptomatic trephining, and that by these means also the excessive use of morphia can be avoided.

STANLEY BARNES.

### THE USE OF ACID PHOSPHATE OF SODIUM IN ALKALINITY

(36) **OF THE URINE.** ROBERT HUTCHISON, *Brit. Med. Journ.*, May 1903, p. 1256.

DR HUTCHISON has made a series of experiments on the influence of different drugs upon the reaction of the urine. After obtaining a standard of normal acidity, various substances were administered, and the effect upon the acidity of the urine observed. The influence of mineral acids (hydrochloric, sulphuric, phosphoric) was extremely slight. Organic acids (tartaric, acetic, citric and lactic) had a slight but distinct tendency to increase the acidity of the urine. Benzoic and boric acids had no influence on the reaction of the urine.

Acid salts were then tried, sodium acid phosphate being chosen. This salt, which is the chief cause of the acid reaction of normal urine, "had a more direct and marked influence on the reaction of the urine than any other substance tried." This led the author to try the effect of acid sodium phosphate in cases of alkaline urine. He has treated many cases with acid phosphate of sodium, and "the power of the drug to increase the acidity of the urine is quite manifest. . . . The drug is very soluble in water. . . . It may be given in doses of from 30 to 60 grains every three hours. . . . A convenient plan is to dissolve 2 drachms in a pint of water and allow the patient to drink small quantities from time to time."

Diarrhoea may be produced, but is usually easily checked. The drug may be given together with urotropin, which acts best in an acid urine.

EDWIN BRAMWELL.

### THREE CASES OF TUMOUR INVOLVING THE SPINAL CORD

(37) **TREATED BY OPERATION.** J. J. PUTNAM and J. W.

ELLIOTT, *Journ. Nerv. and Ment. Dis.*, Nov. 1903.

THE first case recorded is that of a man who, after two accidents apparently attended with spinal injury, developed motor and sensory paralysis of all four extremities; paralysis of the diaphragm threatening, he was operated upon and an infiltrating giant-celled sarcoma removed from the extra-dural space around the second cervical segment. The growth had eroded the axis, and almost encircled the dura at this point, the dura being infiltrated with the disease. After the operation, the patient slowly but steadily recovered, and when examined five years later he could get about and follow his occupation. There was no sign of recurrence.

The second case was that of a patient with symptoms pointing to a secondary carcinomatous growth compressing the cord in the lower dorsal region, and causing paraplegia and intense pain. The growth was removed as far as possible by operation, and the posterior nerve-roots involved were cut, the dura not being opened. The operation greatly relieved the pain, but she died a few months later of recurrence.

The third case was that of a man aged 54, with well-marked signs of compression of the spinal cord in the lower cervical region. Operation revealed a rather diffuse fibro-sarcoma, four inches in vertical extent, chiefly extra-dural, but so adherent to the theca that a piece of the latter was removed with the growth; much cerebro-spinal fluid escaped at the operation. The patient died two days later, the temperature rising to 108° F.

The authors comment upon these cases, particularly calling attention to—

- (a) The non-recurrence of a sarcomatous growth after five years;
- (b) The value of a palliative operation in malignant growths of the spine to alleviate pain; and
- (c) The rapid occurrence of death with high temperature after opening the dura in certain cases.

There is no post-mortem account of the third case.

STANLEY BARNES.

# Bibliography

## ANATOMY

- HIS. Die Entwicklung des menschlichen Gehirns während der ersten 4 Monate des Intrauterinen Lebens. *Hirzel*, Leipzig, 1903, M. 14.
- JOHN TURNER. Notes on the Minute Structure of the Human Caudate Nucleus and Optic Thalamus. *Brain*, Autumn, 1903, p. 400.
- SERGE SOUKHANOFF. On the Intracellular Network of Golgi of the Nervous Elements of the Spinal Cord in the Adult Superior Vertebrate. *Journ. Ment. Pathol.*, vol. v., No. 1, 1903, p. 1.
- BOYCOTT. On the Number of Modes of Ranvier in Different Stages of the Growth of Nerve Fibres in the Frog. *Journ. of Physiol.*, Dec. 1903, p. 370.
- WEIGNER. Ein Beitrag zur Bedeutung des Gehirngewichts beim Menschen. *Anat. Hefte*, Bd. 23, H. 1, 1903, S. 67.
- LANGLEY. On the Sympathetic System of Birds, and on the Muscles which Move the Feathers. *Journ. of Physiol.*, Dec. 1903, p. 221.

## PHYSIOLOGY

- DURANTE. Le Neurone et ses impossibilités. Conception caténaire du tube nerveux et son rôle actif dans la transmission nerveuse. *Rev. Neurol.*, Nov. 30, 1903, p. 1089.
- KURT GOLDSTEIN. Die Zusammensetzung der Rückenmarkshinterstränge. *Monatsschr. f. Psychiat. u. Neurol.*, Bd. 14, H. 6, 1903, S. 401.
- KÖSTER. Ueber die verschiedene biologische Werthigkeit der hinteren Wurzel und des sensiblen peripheren Nerven. Vorläufige Mittheilung. *Neurol. Centralbl.*, Dec. 1, 1903, S. 1093.
- LEWANDOWSKY und SCHULTZ. Ueber Durchschneidungen der Blasen-nerven. *Zentralbl. f. Physiol.*, Bd. 17, Nr. 16, 1903, S. 433.
- VON OPPOLZER. Grundzüge einer Farbentheorie. II. *Ztschr. f. Psychol. u. Physiol. d. Sinnesorgane*, Bd. 33, H. 5, 1903, S. 321.
- E. YUNG. Recherches sur le sens olfactif de l'escargot. *Arch. de Psychol.*, T. iii., No. 9, 1903, p. 1.
- HUGO FREY. Weitere Untersuchungen über die Schalleitung im Schädel. *Ztschr. f. Psychol. u. Physiol. d. Sinnesorgane*, Bd. 33, H. 5, 1903, S. 355.
- SEMENOFF. Ueber die functionellen Veränderungen des Nerven unter dem Einfluss der mechanischen Compression. *Arch. f. d. ges. Physiol.*, Bd. 100, H. 1-4, 1903, S. 182.
- PAERNA. Functionelle Veränderungen der Nerven in Elektrotonus. *Arch. f. d. ges. Physiol.*, Bd. 100, H. 1-4, 1903, S. 145.
- BOIGEY. Rapports entre l'activité cérébrale et la composition des urines. *Arch. gén. de Méd.*, Dec. 15, 1903, p. 3146.
- UCHTOMSKY. Ueber den Einfluss der Anämie auf das Nerven-Muskel-apparat. *Arch. f. d. ges. Physiol.*, Bd. 100, H. 1-4, 1903, S. 190.
- JENSEN. Die physiologischen Wirkung des Lichtes. *Wien. med. Wchnschr.*, Dec. 5, 1903, S. 2322.
- D'ORMEA. Sur les modifications de la circulation cérébrale à la suite de l'administration de quelques essences. *Arch. ital. de biol.*, T. xv., f. 1, 1903, p. 141.

## PSYCHOLOGY

- McKEEN CATTELL. Statistics of American Psychologists. *Am. Journ. Psychol.*, July-Oct. 1903, p. 310.
- BUCHNER. A Quarter Century of Psychology in America. *Am. Journ. Psychol.*, July-Oct. 1903, p. 402.
- FREDERICK R. FARMER. A Scheme of Brain Storage. With some New Theories connected with Environment. Kegan, Paul, Trench & Co., London, 1903, 2s.

- YUJIRO MOTORA. A Study on the Conductivity of the Nervous System. *Am. Journ. Psychol.*, July-Oct., 1903, p. 329.
- STODDART. The Evolution of Consciousness. *Brain*, Autumn, 1903, p. 432.
- JOSEPH JASTROW. The Status of the Sub-conscious. *Am. Journ. Psychol.*, July-Oct. 1903, p. 79.
- ADOLPH MEYER. An Attempt at Analysis of the Neurotic Constitution. *Am. Journ. Psychol.*, July-Oct. 1903, p. 90.
- CLAPAREDE. Le mental et le physique d'après L. Busse. *Arch. de Psychol.*, T. III, No. 9, 1903, p. 81.
- PAUL SCHULZ. Gehirn und Seele. Barth, Leipzig, 1903. 2s.
- MONTROSE WHIPPLE. Studies in Pitch Discrimination. *Am. Journ. Psychol.*, July-Oct. 1903, p. 289.
- KIRSCHMANN. Deception and Reality. *Am. Journ. Psychol.*, July-Oct. 1903, p. 24.
- HYSLOP. Binocular Vision and the Problem of Knowledge. *Am. Journ. Psychol.*, July-Oct. 1903, p. 42.
- MADISON BENTLEY. A Critique of "Fusion." *Am. Journ. Psychol.*, July-Oct. 1903, p. 60.
- MARGARET WASHBURN. The Genetic Function of Movements of Organic Sensations for Social Consciousness. *Am. Journ. Psychol.*, July-Oct. 1903, p. 73.
- PATRICK. The Psychology of Football. *Am. Journ. Psychol.*, July-Oct. 1903, p. 104.
- BURNHAM. Retroactive Amnesia. Illustrative Cases and a Tentative Explanation. *Am. Journ. Psychol.*, July-Oct. 1903, p. 118.
- CHAMBERLAIN. Primitive Taste-words. *Am. Journ. Psychol.*, July-Oct. 1903, p. 146.
- BEATRICE EDGEELL. On Time Judgments. *Am. Journ. Psychol.*, July-Oct. 1903, p. 154.
- TITCHENER. Class Experiments and Demonstration Apparatus. *Am. Journ. Psychol.*, July-Oct. 1903, p. 176.
- MAX MEYER. Experimental Studies in the Psychology of Music. *Am. Journ. Psychol.*, July-Oct. 1903, p. 192.
- KNEPPE. Ein Beitrag zur Experimentelle Aesthetik. *Am. Journ. Psychol.*, July-Oct. 1903, p. 215.
- BOLTON. The Relation of Motor Power to Intelligence. *Am. Journ. Psychol.*, July-Oct. 1903, p. 361.
- DRESSLAR. Are Chromaesthesias variable? A Study of an Individual Case. *Am. Journ. Psychol.*, July-Oct. 1903, p. 368.
- SANFORD. On the Guessing of Numbers. *Am. Journ. Psychol.*, July-Oct. 1903, p. 383.
- TERRIER. Psychologie physiologique: Le chimpanzé "Consul." *Gaz. méd. de Paris*, Nov. 1903, p. 413.
- LANUSSE. Une question de psychologie critique. Suer-Charruey, Paris, 1903.
- LENBA. The State of Death: an Instance of Internal Adaptation. *Am. Journ. Psychol.*, July-Oct. 1903, p. 183.
- BEAUNIS. Contribution à la psychologie du Rêve. *Am. Journ. Psychol.*, July-Oct. 1903, p. 7.
- BERGSTROM. A New Type of Ergograph with a Discussion of Ergographic Experimentation. *Am. Journ. Psychol.*, July-Oct. 1903, p. 246.
- CASWELL ELLIS and MAUD SHIPE. A Study of the Accuracy of the Present Methods of Testing Fatigue. *Am. Journ. Psychol.*, July-Oct. 1903, p. 232.
- ALTOBELLI. Recherches sur le rapport entre visus et lumière (Influence de la fatigue). *Arch. ital. de Biol.*, T. xv, f. 1, 1903, p. 99.
- PILLSBURY. Attention Waves as a Means of Measuring Fatigue. *Am. Journ. Psychol.*, July-Oct. 1903, p. 277.

## PATHOLOGY

- HENRI CARRIER. La Cellule Nerveuse normale et pathologique. Baillière et fils, Paris, 1903. 7 fr.
- G. ALFRED LAWRENCE. Studies upon the Cerebral Cortex in the Normal Human Brain and in Dementia Paralytica. *Journ. Nerv. and Ment. Dis.*, Dec. 1903, p. 754.
- LEVINSOHN. Ueber das Verhalten des Ganglion cervicale supremum nach Durchschneidung seiner prae. bzw. postocellulären Fasern. *Arch. f. Anat. u. Physiol.*, H. 5 u. 6, 1903, p. 438.

- FRANCHETTI. Sulle cellule giganti da corpi estranei. *Sperimentale*, Anno LVII., f. 5, 1903, p. 607.
- MAINZER. Stoffwechselstudien über den Einfluss geistiger Tätigkeit und protrahierten Wachens. *Monatsschr. f. Psychiat. u. Neurol.*, Bd. 14, H. 6, 1903, S. 442.
- HAUSHALTER et RICHON. Malformation cardiaque et cavité médullaire chez un enfant de 10 mois. *Rev. mem. d. mal. de l'enfance*, Dec. 1903, p. 562.
- BRAEUNIG. Ueber Degenerationsvorgänge im Organismus bei der Transfusion fremdartigen Blutes. *Arch. f. Anat. u. Physiol.*, H. 5 u. 6, 1903, p. 494.
- HILL and MACLEOD. Caisson Illness and Diver's Palsy. An Experimental Study. *Journ. of Hygiene*, vol. iii., No. 4, 1903, p. 401.
- D. PACE. Della natura e significato delle lesioni istologiche nelle cicatrici rabiche dell' uomo. *Ann. di Neurolog.*, f. 3, 4, 1903.

## CLINICAL NEUROLOGY AND PSYCHIATRY

### GENERAL—

- SIR WILLIAM BROADBENT. Hughlings Jackson as Pioneer in Nervous Physiology and Pathology. *Brain*, Autumn, 1903, p. 305.
- HOLST. Erfahrungen aus vierzigjährigen neurologischen Praxis. F. Enke, Stuttgart, 1903, M. 1.60.
- LUCIEN PRON. Influence de l'estomac sur l'état mental et les fonctions psychiques. Rousset, Paris, 1904, 3 fr.
- J. MONTGOMERY MOSHER. Electrodiagnosis. Scheme for the Differential Testing of Nerves and Muscles. Albany, 1903.

### PERIPHERAL NERVES—

- LE BRETON. Contribution à l'histoire des névrites périphériques chez les tuberculeux (forme paralytique). Michalon, Paris, 1903.
- R. COLELLA. Studio sulla polinevrite tubercolare. *Ann. di Neurolog.*, f. 2, 1903.
- REMSEN. A Case of Syphilis of the Peripheral Nerves and Lungs. *Bull. of the Johns Hopkins Hospital*, vol. xiv., No. 151, 1903, p. 280.
- BABONNEIX. Les paralysies diphtériques et la théorie de la méningite. *Gaz. des hôp.*, Dec. 15, 1903, p. 1429.
- R. E. F. SQUIBBS. Notes of a Case of Paralysis of the Upper Extremities probably due to Diphtheria, accompanied by a Rash (Septic). *Lancet*, Nov. 21, 1903, p. 1481.
- LENTHAL CHEATLE. The Behaviour of Carcinoma within Nerve and Trophic Areas. *Brit. Med. Journ.*, Dec. 12, 1903, p. 1515.

### SPINAL CORD—

- Tabes.**—FLATAU. Die Tabes dorsalis (Rückenmarkschwindtsucht). Konagen, Leipzig, 1903, M. 1.50.
- ALLAN M. HAMILTON. Traumatic Locomotor Ataxia. *Med. Rec.*, Nov. 21, 1903, p. 801.
- SCHULZE. Ueber Psychoosen bei Tabes. *Munch. med. Wchnschr.*, Dec. 8, 1903, S. 2181.
- PAL. Ueber Gefätskrisen und deren Beziehung zu den Magen- und Bauchkrisen der Tabetiker. *Munch. med. Wchnschr.*, Dec. 8, 1903, S. 2185.
- CURIONI. Rapports du tabes avec la paralysie générale. *Nouv. Icon. de la Salpêtrière*, Sept.-Oct. 1903, p. 276.
- JOFFROY et RABAUD. Un cas d'association du tabes à la paralysie générale. *Rev. Neurol.*, Nov. 30, 1903, p. 1081.
- CHOMPRET. Mal perforant buccal ou nécroses multiples du maxillaire chez un tabétique. *Arch. gén. de méd.*, Dec. 1, 1903, p. 8009.
- E. DE MASSARY. La nature syphilitique et la curabilité du tabes et de la paralysie générale. *Arch. gén. de méd.*, Nov. 24, 1903, p. 2959.
- Paraplegia.**—BALLET et LAIGNEL-LAVASTINE. Deux cas de myélite aiguë au cours d'un carcinome secondaire de la dure-mère ou de la moelle. *Rev. Neurol.*, Dec. 15, 1903, p. 1129.
- CAMPBELL THOMSON. Hereditary Spastic Paraplegia. Its Relation to Friedrich's Disease and its Claim to be Considered as a Clinical Entity. *Brain*, Autumn, 1903, p. 412.



- Myelitis.**—W. G. SPILLER. A Report of Two Cases of Paraplegia occurring in Variola, one being a Case of Anterior Poliomyelitis in an Adult. *Brain*, Autumn, 1903, p. 424.
- Fracture of Spine.**—E. F. TINLEY and A. R. JONES. Fracture of the Fifth Cervical Vertebra; Recovery. *Lancet*, Nov. 28, 1903, p. 1498.
- Syringomyelia.**—BORCHARD. Die Verbiegung der Wirbelsäule bei der Syringomyelie. *Mittheil. a. d. Grenzgebieten d. Med. u. Chir.*, Bd. 12, H. 4, 1903, S. 483.
- Disseminated Sclerosis.**—RAMSAY HUNT. Multiple Sclerosis with Dementia. *Am. Journ. Med. Sc.*, Dec. 1903, p. 974.
- BYROM BRAMWELL. Ataxic Paraplegia and Spastic Paraplegia as Symptoms of Disseminated Sclerosis. *Rev. Neurol. and Psychiat.*, Dec. 1903, p. 791.
- Tumour of Cauda Equina.**—CHARLES R. BOX. A Case of Invasion of the Cauda Equina by Tumour, with Demarcation of all the Sensory Root Areas of the Lower Limbs. *Lancet*, Dec. 5, 1903, p. 1566.

# BRAIN AND MEMBRANES—

- WM. CHARLES WHITE. Lesion of the Brachium Pontis, with Division of the Trigeminal and Facial Nerves. *Brain*, Autumn, 1903, p. 440.
- Hydrocephalus.**—LANGSTEIN. Zur Kenntnis der Cerebrospinalflüssigkeit in einem Fall von chronischem Hydrocephalus. *Jahrb. f. Kinderk.*, Dec. 1, 1903, S. 926.
- Meningitis.**—LAIR. Éléments de diagnostic et de pronostic dans les méningites cérébro-spinales. Thèse, Paris, 1903.
- BARTHELEMY. Sur un cas de méningite cérébro-spinale. *Gaz. méd. de Nantes*, Oct. 31, 1903, p. 45.
- SERGENT et LEMAIRE. Méningite cérébro-spinale à staphylocoque chez deux typhiques. Contagiosité possible. *Arch. gén. de méd.*, Dec. 8, 1903, p. 3073.
- H. LAMY. Hémorragie méningée traumatique. Syndrome méningitique. Guérison rapide. *Soc. méd. des hôp.*, Nov. 5, 1903.
- CHAOFFARD et FROIN. Du diagnostic différentiel de l'hémorragie méningée sous-arachnoidienne, et de la méningite cérébro-spinale. *Soc. méd. des hôp.*, Oct. 29, 1903, p. 1108.
- ACHARD et GRENET. Hémorragie méningée dans le cours d'une méningite cérébro-spinale. *Soc. méd. des hôp.*, Nov. 5, 1903, p. 1121.
- MALJEAN. Méningite cérébro-spinale aiguë consécutive à une otite moyenne grippale. Guérison complète par les ponctions lombaires. *Ann. mal. du larynx*, Oct. 1903, p. 340.
- GAY. Du sérum antidiphtérique dans la pneumonie et la méningite infectieuse. *Gaz. degli ospedali*, Oct. 11, 1903, p. 1287.
- TRÉMOLIERES. Le diagnostic de la méningite tuberculeuse par les procédés nouveaux. *Gaz. des hôp.*, Nov. 7, 1903, 1269.
- BAUER. Hémorragie méningée sous-arachnoidienne par lésion du tronc basilaire. Ponction lombaire. Autopsie. *Arch. gén. de méd.*, Déc. 1, 1903, p. 3026.
- STARCK. Hemiplegia superior alternans bei Meningitis tuberculosa. *Beitr. z. Klinik d. Tuberkulose*, Bd. 11, H. 1, 1903, S. 73.
- Lumbar Puncture.**—BUSCHMANN. Meningitis und Lumbalpunktion. Speyer und Kaerner, Freiburg, 1903, M. 1.
- CHAVASSE and MAHA. La ponction lombaire dans les complications endocraniennes des otites. *Presse méd.*, Nov. 25, 1903, p. 815.
- DONATH. Beiträge zum diagnostischen und therapeutischen Wert der Quincke'schen Lumbalpunktion. *Wien. med. Wchnschr.*, Dec. 5, 1903, S. 2306.
- A. SIGNORELLI. Contribution à l'étude cytologique des épanchements liquides inflammatoires des différents séreuses et spécialement du liquide céphalo-rachidien. *Arch. latines de méd. et biol.*, No. 1, 1903.
- Cerebral Embolus.**—WALTER BROADBENT. Cerebral Embolus during an Epileptic Fit. *Brain*, Autumn, 1903, p. 447.
- Intracranial Tumours.**—DURET. Sur les manifestations des tumeurs du lobe occipital et du lobe temporopariétal. *Rev. Neurol.*, Nov. 15, 1903, p. 1029.
- PAVIOT. Tumeurs du IV<sup>e</sup> ventricule avec vomissements incoercibles et fécaloïdes. *Rev. Neurol.*, Nov. 15, 1903, p. 1033.
- F. X. DERCUM. Enormous Tumour of the Postero-Parietal Region Weighing over half a pound; Absence of Localised Symptoms until late in the History of the Case; Operation; Death. *Journ. Nerv. and Ment. Dis.*, Dec. 1903, p. 737.
- FERRARINI e PAOLI. Glioma bilaterale del nucleo lentiforme e miotonia. *Ann. di Neurolog.*, f. 3, 4, 1903.
- WOOLSEY. A Contribution to the Surgery of Cerebral Tumours. *Am. Journ. Med. Sc.*, Dec. 1903, p. 956.

- Cerebellar Abscess.**—OPPENHEIMER. Mastoid Diseases and Cerebellar Abscess. *Med. News*, Nov. 28, 1903, S. 1011.
- General Paralysis.**—A. MARIE ET VIOLETT. Traumatismes multiples et paralysie générale. *Prog. méd.*, Nov. 14, 1903, p. 401.
- DIEFENDORF. The Blood Changes in Dementia Paralytica. *Am. Journ. Med. Sc.*, Dec. 1903, p. 1047.
- WEBER. Ueber die sog. gallopiierende Paralyse nebst einigen Bemerkungen über Symptomatologie und patholog. Anatomie dieser Erkrankung. *Monatsschr. f. Psychiat. u. Neurol.*, Bd. 14, H. 6, 1903, S. 450.
- Pellagra.**—MORICHAN-BEAUCHANT. Un cas de pellagre indigène. *Gaz. des hôp.*, Dec. 1, 1903, p. 1369.
- GRAXA. Contributo alle cognizioni sull' etiologia della pellagra. *Ann. d'Igiene Speriment.*, vol. xiii, f. 3, 1903, p. 367.

#### GENERAL AND FUNCTIONAL DISEASES—

- Epilepsy.**—THEODOR ZAHN. Eine merkwürdige Gedächtnisleistung in einem epileptischen Dämmerzustande. *Allg. Ztschr. f. Psychiat.*, Bd. 16, H. 6, 1903, S. 889.
- MEYER. Zur diätetischen Brombehandlung der Epilepsie. *Berl. klin. Wchnschr.*, Nov. 16, 1903, S. 1049.
- WILDERMUTH. Über die Aufgaben des Pflegepersonals bei Epileptischen. Marhold, Halle, 1904, M. 0.50.
- Neurasthenia.**—LE CLECH. Le neurasthénie grave d'origine osseuse. *Impr. nationale*, Paris, 1903.
- NEUMANN. Die Heilung der Nervosität durch intelligente Leibes- und rationelle Lebenshaltung. Borggold, Leipzig, 1903, M. 1.20.
- Hysteria.**—THIEMICH. Ueber Hysterie im Kindesalter. *Jahrb. f. Kinderh.*, Dec. 1, 1903, S. 881.
- BRUNS. Die Hysterie im Kindesalter. *Jahrb. f. Kinderh.*, Dec. 1, 1903, S. 895.
- LÉVI et MALLOZEL. Hystéro-traumatisme ou foyers hémorragiques avec hémorragie principale de la protubérance. *Rev. Neurol.*, Déc. 15, 1903, p. 1139.
- PAGLIANO et AUDIBERT. Quadriplégie hystérique à forme de paralysie périodique familiale. *Arch. gén. de méd.*, Dec. 8, 1903, p. 3081.
- CARNOT et LAMOUROUX. Sur un cas de pseudo-paralysie saturnine d'origine hystérique. *Soc. méd. des hôp.*, Nov. 5, 1903, p. 1903.
- Tic.**—MEIGE. Les tics des yeux. *Doyn, Paris*, 1903.
- Neuralgia.**—FRAZIER. Clinical Lecture on the Symptomatology and Treatment of Trifacial Neuralgia. *Am. Journ. Med. Sc.*, Dec. 1903, p. 1005.
- Tetanus.**—FLETCHER. Tetanus Dolorosus, and the Relation of Tetanus Toxin to the Sensory Nerves and the Spinal Ganglia. *Brain*, Autumn, 1903, p. 383.
- Tetany.**—ESCHERICH. Die tetanoiden Erkrankungen des ersten Kindesalters. *Wien. med. Presse*, Dec. 13, 1903, S. 2360.
- Thomson's Diseases.**—JAQUET. Les troubles de la motilité dans la maladie de Thomsen. *Semaine méd.*, Nov. 25, 1903, p. 381.
- Myasthenia Gravis.**—MOHR. Ein Beitrag zur myasthenischen Paralyse. *Berl. klin. Wchnschr.*, Nov. 16, 1903, S. 1052.
- Exophthalmic Goitre.**—TAUBMANN. Les états psychiques morbides et les vésanies dans la maladie de Basedow. Thèse, Paris, 1903.
- Dercum's Disease.**—DEBOVE. L'adipose douloureuse, ou maladie de Dercum. *Arch. gén. de Méd.*, Dec. 15, 1903, p. 1516.
- DENY et LE PLAY. Adipose sous-cutanée symétrique et segmentaire chez une démente alcoolique et hérédito-alcoolique. *Nouv. Icon. de la Salpêtrière*, Sept.-Oct. 1903, p. 230.

#### MENTAL DISEASES—

- R. SOMMER. Die Classification der Psychoosen. *Beitr. z. psychiat. Klinik*, Bd. 1, H. 4, 1903, S. 183.
- T. S. CLOUSTON. The Prodromata of the Psychooses, and their Meaning. *Rev. Neurol. and Psychiat.*, Dec. 1903, p. 781.
- DAMAYE. Essai de diagnostic entre les états de débilites mentales. Steinheil, Paris, 1903.
- MEURIOT. Des hallucinations des obsédés, Pseudo-hallucinations. Corbell, Crété, 1903.
- GRIMALDI. L'origine affettiva dei delirii paranoici nella letteratura tedesca. *Ann. di Neurolog.*, f. 3, 4, 1903.

- A. DI LUZENBERGER. A proposito dell' Ereutofobia. Contributo allo studio del meconismo delle idee fisse e considerazioni psicologiche. *Ann. di Neurolog.*, f. 3, 4, 1903.
- NINA RODRIGUES. La paranoia chez les nègres. *Arch. d'Anthrop. Crim.*, T. xviii, No. 119, 1903, p. 639.
- PILCZ. Beiträge zur Klinik der periodischen Psychoeen. *Monatsschr. f. Psychiat. u. Neurol.*, Bd. 14, H. 6, 1903, S. 434.
- RÜDIN. Eine Form akuten halluzinatorischen Verfolgungswahns in der Haft ohne spätere Weiterbildung des Wahns und ohne Korrektur. *Allg. Ztschr. f. Psychiat.*, Bd. 16, H. 6, 1903, S. 852.
- R. SOMMER. Zur Kritik des Dementia praecox. *Beitr. z. psychiat. Klinik*, Bd. 1, H. 4, 1903, S. 182.
- PAUL MASOIN et HENRY MEIGE. Les possédés de l'église Sainte-Dymphne. *Nouv. Icon. de la Salpêtrière*, Sept.-Oct. 1903, p. 305.
- A. MARIE. Possédés guéris par les reliques de Saint Etienne, d'après une tapisserie berrichonne du XVe siècle. *Nouv. Icon. de la Salpêtrière*, Sept.-Oct. 1903, p. 302.
- CAPGRAS. Contribution à l'étude de la névrose d'angoisse. *Ann. méd.-psychol.*, nov.-déc., 1903, p. 397.
- Criminal Psychiatry.**—ROBINOVITCH. Suicidal and Homocidal Acts; their Criminal Aspects and Medico-legal Significance. *Journ. Ment. Pathol.*, vol. v., No. 1, 1903, p. 7.
- KOEPPEN. Sammlung von Gerichtlichen Gutachten aus der Psychiatrischen Klinik der Königl. Charité zu Berlin. S. Karger, Berlin, 1904, 15a. 6d.
- MARIANI. La degenerazione criminosa nella discendenza degli alienati. *Riv. mens. di Psichiat. forense*, Anno VI., No. 10, 1903, p. 405.
- Treatment.**—STEGMANN. Die Grenzen der Verwendbarkeit hypnotischen Schlags in der Psychotherapie. *Munch. med. Wchnschr.*, Dec. 8, 1903, S. 2141.
- WEYGANDT. Die Fürsorge für schwachsinnige Kinder in Bayern. *Allg. Ztschr. f. Psychiat.*, Bd. 16, H. 6, 1903, S. 833.
- SCHLOSS. Pflege und Behandlung von zu Geistesstörungen disponierten Kindern. *Wien. med. Wchnschr.*, Dec. 12, 1903, S. 2354.
- MARTINET. La medication phosphorique dans la psychasténie. *Presse méd.*, Nov. 21, 1903, p. 805.

#### ALCOHOLISM—

- HIRT. Nervensystem und Alkohol. Bergmann, Wiesbaden, 1903, M. 1.
- MARCHIAFAVA e BIGNAMI. Sopra un'alterazione del corpo calloso osservata in soggetti alcoolisti. *Riv. di Patolog. nerv. e ment.*, vol. viii., f. 12, 1902, p. 544.
- DEROUBAIX. Syndrome de Korsakoff et paralysie générale. *Journ. de Neurol.*, Nov. 20, 1903, p. 537.
- SOUKHANOFF et POUTENKO. Sur le diagnostic différentiel de la maladie de Korsakoff. *Journ. de Neurol.*, Nov. and Dec., 1903, pp. 515, 535.
- S. B. ELLIOT. Restraint and Moral Measures in the Treatment of Inebriety. *Quart. Journ. Inebriety*, No. 25, 1903, p. 24.
- A. RHU. A Plea for Early Treatment of the Alcohol, Morphine, Cocaine, and Allied Narcotic Drug Habits. *Am. Med. Compend.*, No. 19, 1903, p. 25.
- J. H. KELLOGG. The Treatment of Drug Addiction. *Quart. Journ. of Inebriety*, No. 25, 1903, p. 30.

#### CRANIAL NERVES—

- TAKABATAKE. Die Veränderungen an den Sehnervenseiben bei den otogenen Erkrankungen des Hirns der Hirnhäute und der Blutleiter. *Ztschr. f. Ohrenh.*, Bd. 45, H. 3, 1903, S. 224.
- ÉMILÉ BERGER et ROBERT LOEWY. Sur la contracture secondaire du releveur de la paupière supérieure dans le cours de la paralysie faciale. *Rev. Neurol.*, Dec. 15, 1903, p. 1144.
- LÉPINE. Deux cas de tumeur du nerf auditif. *Rev. Neurol.*, Nov. 30, 1903, p. 1104.
- Pupil.**—THOMAS. The Anatomical Basis of the Argyll-Robertson Pupil. *Am. Journ. Med. Sc.*, Dec. 1903, p. 939.
- BACH. Ueber die reflectorische Pupillenstarre und den Hirnrindenreflex der Pupille. *Neurol. Centralbl.*, Dec. 1, 1903, S. 1090.
- BUMKE. Beiträge zur Kenntnis der Iriabewegungen. *Centralbl. f. Nervenh. u. Psychiat.*, Nov. 15, 1903, S. 673.

- H. K. ANDERSON. The Paralysis of Involuntary Muscle, with Special Reference to the Occurrence of Paradoxical Contraction. Part I. Paradoxical Pupil-Dilatation and other Ocular Phenomena caused by Lesions of the Cervical Sympathetic Tract. *Journ. of Physiol.*, Dec. 1903, p. 290.
- LEVINSOHN. Ueber das Verhalten der Irmuskeln bei traumatischer Pupillen-  
klümmung. *Klin. Monatsbl. f. Augenh.*, Bd. 41, H. 2, 1903, S. 515.
- HARNIS. Einseitige spontane Tückenbildung der Iris durch Atrophie ohne  
mechanische Zerrung. *Klin. Monatsbl. f. Augenh.*, Bd. 41, H. 2, 1903, S. 522.

## REFLEXES—

- GOLDFLAM. Zur Lehre von den Hautreflexen an den Unterextremitäten  
(insbesondere des Babinaki'schen Reflexes). *Neurol. Centralbl.*, Dec. 1 and 16,  
1903, S. 1109. u. 1137.
- BYROM BRAMWELL. Note on the Crossed Plantar Reflex. *Journ. Nerv. and  
Ment. Dis.*, Dec. 1903, p. 795.

## MISCELLANEOUS SYMPTOMS—

- KNAPP. Ein Fall von akut aufgetretener reiner Tastklümmung. *Monatschr. f.  
Psychiat. u. Neurol.*, Bd. 14, H. 6, 1903, S. 428.
- HÁSKOVEC. Nouvelles remarques sur l'akathisie. *Nouv. Icon. de la Salpêtrière*,  
Sept.-Oct. 1903, p. 287.
- GAUPP. Zur prognostischen Bedeutung der katatonischen Erscheinungen.  
*Centralbl. f. Nervenh. u. Psychiat.*, Nov. 15, 1903, S. 630.
- Respiratory Movement.**—MOSSO. Les mouvements respiratoires du thorax et  
du diaphragme. *Arch. ital. de Biol.*, T. xv., f. 1, 1903, p. 43.
- PIERCE CLERK. The Movements of Superior Intercostal Muscles in Hemi-  
plegics. *Ann. Journ. Med. Sc.*, Dec. 1903, p. 1029.
- JUDSON S. BURY. Note on the Respiratory Movements in Hemiplegia.  
*Lancet*, Dec. 19, 1903, p. 1714.
- Speech.**—HAMMERSCHLAG. Beitrag zur Lehre von den Sprachstörungen im  
Kindesalter. *Ztschr. f. Ohrenh.*, Bd. 45, H. 3, 1903, S. 254.
- G. SAINT-PAUL. Le langage intérieur et les paraphasies (la fonction en-  
doplasique). Félix Alcan, Paris, 1903.
- QUENSEL. Zur Pathologie der amnestischen Aphasie. *Neurol. Centralbl.*,  
Dec. 1, 1903, S. 1102.
- LEMAITRE. Des phénomènes de paramnésie. *Arch. de Psychol.*, T. iii., Nov. 9,  
1903, p. 101.

## TREATMENT—

- Surgical.**—WITEK. Die Bedeutung der Sehnentransplantation für die Behand-  
lung choreatischen Formen der infantilen Cerebralklümmung. *Mittheil. a. d.  
Grenzgebieten d. Med. u. Chir.*, Bd. 41, H. 4, 1903, S. 498.
- FARRER COBB. Report of a Case of Recurrent Dislocation of the Ulnar Nerve  
cured by Operation. *Ann. Surg.*, Nov. 1903, p. 652.
- OSCAR VULPIUS. Der heutige Stand der Sehnenplastik. *Wien. Klinik*, Bd.  
29, H. 12, 1903, S. 339.
- HENDERSON NICOLL. The Operative Treatment of Cranial Depression  
(Greenstick Fracture of the Skull). *Brit. Med. Journ.*, Dec. 19, 1903, p. 1578.
- Electrical.**—KURELLA. Elektrotherapie und Wirtschaft. *Ztschr. f. Elektrother.  
u. physikal. Heilmethoden*, Bd. 5, H. 10, 1903, S. 319.
- STÉPHANE LÉDUC. La Narcoose Électrique. *Ztschr. f. Elektrother. u. physikal.  
Heilmethoden*, Bd. 5, H. 11, 1903, S. 374.
- FOVEAU DE COURMELLES. Des diverses lumières et de leurs actions théra-  
peutiques. *Ztschr. f. Elektrother. u. physikal. Heilmethoden*, Bd. 5, H. 11, 1903,  
S. 382.
- LOEWY und NEUMANN. Erfahrungen über die elektromagnetische Therapie.  
*Ztschr. f. Elektrother. u. physikal. Heilmethoden*, Bd. 5, H. 11, 1903, S. 359.

# Review

of

## Neurology and Psychiatry

---

### Original Articles

#### **A CASE OF PRIMARY SCLEROSIS OF THE POSTERIOR COLUMNS, FOLLOWED BY DISSEMINATED SOFTENING OF THE OTHER WHITE COLUMNS.** **A Contribution to the Study of the Subacute Combined Degenerations of the Spinal Cord.**

By JOSEPH COLLINS, New York.

CASES of nervous disease whose clinical delineation does not conform to any recognised type, as well as cases that offer great difficulty of diagnosis, should be recorded in the hope that some day generalisations from them may be possible. It is for this reason that I call attention to the following history, and the findings of the post-mortem investigation.

J. H., a married German, 42 years old, had worked as a house-painter upwards of 25 years. The only illness that he had had, aside from an attack of pneumonia in early manhood, was colic when he was 29 years old. He does not recall how long the colic lasted, but he thinks it may have been several weeks, for he had to take to bed and call a physician. He had similar attacks when 32 and 38 years old. The two last attacks were not so severe as the first. He says he does not know that they were from lead. There is no history of syphilis, gonorrhœa, or of excessive use of alcoholic drinks. The patient maintains that he was in fairly good health until the year 1900. He then com-

plained of trembling of the hands and of inability to use them because of stiffness. With this there was associated intense paræsthesia, which is described by him as if "the hands were packed in sand." Later he complained of pain in the back and of unwieldiness of the lower extremities. The latter was first noted on going up and down a ladder. About Christmas 1900 the strength of the legs began to diminish rapidly, and at this time he was more or less incapacitated from work, except housework, dish-washing, etc. During the following year his principal complaints were of manual paræsthesia and general weakness. Occasionally he had a "cramp" or band sensation in the abdomen, and a sensation of burning and cold in the soles of the feet. Aside from this there was no pain or disagreeable sensations in the lower extremities. He had no desire for sexual intercourse, although he maintained that he had some capacity. There were no symptoms referable to the bladder or bowels. All this time he was under treatment for chronic lead poisoning, but the weakness progressed. About January 1, 1902, he began to be paraplegic, and since then he has been unable to leave the bed. His lower extremities did not lose the power of voluntary motion; they merely got so weak that he could not stand. He was brought into the City Hospital in March 1902, and my first examination of him was made on the 11th of April. The following is a transcript of the notes made then.

The patient, a man apparently of from 50 to 60 years of age, has the appearance of one suffering from Bright's disease, *i.e.* the mucous membranes are pale, the face has a peculiar waxy appearance, and the eyelids are puffy. He can move the lower extremities, and all the muscles contract voluntarily, but slowly and without vigour. He can flex the thigh and extend the foot simultaneously with considerable strength. When the legs are moved passively there is slight spasticity. The knee-jerks are elicited on both sides, the right jerk is slightly plus, the left minus. The ankle-jerks show relatively a similar change. The lower extremities are considerably wasted, and there is slight puffiness over both tibiae. The Babinski reflex can be elicited on both sides, the right being more active than the left; both are typical. Tactile sensibility is impaired in the lower extremities. This is particularly evident: when a sharp object such as a pin is

drawn over the skin it is readily detected, but when a blunt object is pressed against the skin in any part of the lower extremities he does not feel it. He feels the point of a needle, but not the blunt end. Thermal sensibility is preserved, but recognition of it below the knee is very slow. If he answers at all when hot and cold test-tubes are applied below the knee he does so correctly. Pain sensibility is preserved. The blunting of tactile sensibility extends from the feet up to the middle of the thorax. In the upper extremities he can distinguish the head from the point of a pin with considerable readiness, though there seems to be some blunting of keenness. Deep sensibility, articular and muscular, is entirely lost. The faradic irritability of all the muscles of the lower extremities is preserved, the response being prompt to a moderate current. There is no reaction of degeneration. The gross strength of the upper extremities seems to be fairly well preserved, but he says he cannot use the hands properly, "there isn't the right strength or feeling in them." There is no atrophy of the muscles of the upper extremities and no tremor of the hands. The pupils are equal, responsive to light, and the movements of the eyeballs are not limited. There is no tremor of the face or tongue. The mucous membranes are very pale. The heart, whose dulness is obscured by emphysema, is within the nipple line and smaller than normal. The apical impulse cannot be felt. The first sound is nearly absent, the second sound is accentuated, especially over the aortic. There is no murmur. Emphysema obscures the liver dulness also, but the liver is unquestionably small. A bed sore is developing over the sacrum. The abdomen is so rigid that examination of the viscera is impossible.

#### BLOOD EXAMINATION.

Red cells, 1,027,000, moderate poikilocytosis.

Leucocytes, 7500.

Differential count of leucocytes—

Small mononuclear	.	.	.	.	13½	per cent.
Large	„	.	.	.	4	„
Transitional	.	.	.	.	1	„
Polymorphonuclear	.	.	.	.	80	„
Eosinophiles	.	.	.	.	1½	„
Hæmoglobin	.	.	.	.	60	„

The following additional notes were made April 27th, 1902: There is now complete flaccid paraplegia, and the strength of the upper extremities is much diminished. The knee-jerks are not elicitable. There is a Babinski-jerk on both sides, the left greater than the right. The left lower extremity is oedematous. Bed sores are forming over both heels, shoulders, back and sacrum. The patient is incontinent of urine and fæces. He complains of deep pain in the abdomen and of girdle pressure over thorax.

The patient refuses to eat or to take medicine, and from this time until his death, three weeks later, he persisted in this refusal without offering any explanation. He became taciturn and uncommunicative, and although he would answer questions he never volunteered anything or made requests.

The following is a summary of the principal symptoms related in the order of their occurrence in a house-painter forty-two years old, whose only illness bearing upon his condition had been repeated attacks of lead colic:—

(1) Trembling of the hands and inability to use them, because of a subjective sensation of stiffness, and manual paræsthesia. (2) Pain in the back and unwieldiness of the lower extremities. A sensation as if the legs were wooden. (3) Gradual loss of strength in the legs. (4) Girdle sensation of the abdomen; pedal psychroesthesia and kaumesthesia. (5) Two years after the beginning of these symptoms weakness of the lower extremities increased suddenly, constituting paraplegia. (6) Slight spasticity of the paraplegic extremities. (7) Knee-jerks, both elicitable, right plus, left minus. Three weeks before he died the knee-jerks were absent. (8) Babinski reflex elicitable in both feet, livelier on the right side than the left. (9) Hypoesthesia of the lower extremities and the trunk as far as the middle of the thorax. Deep sensibility profoundly impaired. (10) Bed sores and incontinence of urine a short time before death.

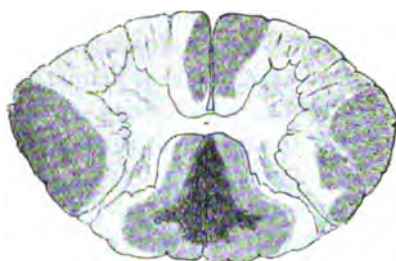
The autopsy was made twenty-four hours after death. The lungs were emphysematous, the heart slightly fatty, the liver small and congested, the kidneys larger than normal, of lighter colour and with indistinct markings. The other abdominal viscera were without noteworthy alterations. The brain and spinal cord showed no abnormality to the naked eye save a thickening of the blood-vessels most perceptible at the base of the brain. There



was an increase of cerebro-spinal fluid. The spinal cord was prepared in the customary way for microscopical study. It showed two distinct varieties of lesions, sclerotic and softening.

1. *Distribution and Nature of the Sclerotic Lesion.*—The most striking alteration is a sclerotic process which confines itself narrowly to the column of Goll and extends from the fourth cervical to the first lumbar segments. Throughout the cervical region, and as far down as the seventh dorsal segment, the distribution of the sclerotic lesion is quite uniform. A description of one trans-section from the cervical cord, and of another from the dorsal cord, is sufficient to give an idea of the topography of this sclerotic tract. At the fourth cervical segment, for instance, the sclerosis involves the entire column of Goll except a narrow symmetrical strip bordering the pia. At the junction of the inner  $\frac{2}{3}$  and outer  $\frac{1}{3}$  of this sclerotic involvement of Goll's column, are symmetrical, wing-like projections extending at right angles into the substance of the column of Burdach. These lateral extensions of the sclerotic tissue into the columns of Burdach lie directly across the pathway of the internal arcuate collaterals of the cervical posterior root axones. This infringement on the column of Burdach is especially significant, as will be pointed out later in connection with the initial symptoms of the disease. Above the fourth cervical segment the sclerotic tract may be traced upward to the nuclei of the posterior columns.

From the 8th cervical to the 7th dorsal segment the wing-like projections of the sclerotic tract disappear. They cease at the level of the 8th cervical segments. From the 8th cervical to the 4th dorsal segment no new feature appears in the sclerotic involvement of the column of Goll, except a V-shaped indenture at the base of the sclerotic triangular area. This V-shaped indenture is composed of normal fibres; each half of the V is symmetrically disposed on either side of the posterior septum; the base lies against the periphery of the cord, and the apex splits the base of the sclerotic Goll's column into two tongue-like processes which extend out to the periphery of the cord. In the 5th, 6th and 7th segments the sclerosis is limited to the column of Goll as before, but its base has retreated inwards from the dorsal periphery about  $\frac{1}{3}$  of the length of the posterior septum. In the 8th and 9th dorsal segments interesting variations in the topo-



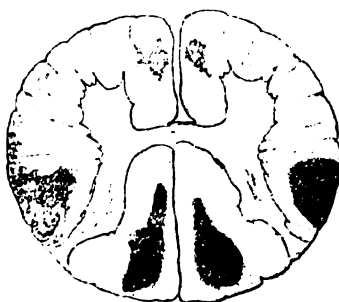
C.vii.



D.v.



D.viii.



L.i.

graphy of the sclerosis begin to appear. The sclerotic area is now no longer strictly confined to the column of Goll, but spreads out, especially at its base, into the column of Burdach, a trifle beyond the boundary line of the interfunicular arteries. If one imagines the basilar truncated sclerotic column of Goll, in the preceding levels, to be split at the base, and the lateral basilar portions thrust asunder on either side of the median line into the columns of Burdach by an indenting triangular mass of normal fibres with the base at the dorsal periphery of the cord, the apex at a point  $\frac{1}{4}$  of the distance inward on the posterior septum, it reproduces the appearance at this level. In the next two segments, 9th and 10th dorsal, there is a continuance and extension of this cleaving of the sclerotic Goll's column into two strips, apexing at a point on the posterior septum at the junction of its inner  $\frac{1}{4}$  and outer  $\frac{3}{4}$ . Thus the apex of the degenerated column of Goll has retreated dorsally from the posterior commissure  $\frac{1}{4}$  of the distance along the posterior septum, while the outer extremities of the strips are quite widely divergent from the median line into

the interfunicular region by the augmentation of the triangular area of normal fibres. From the 11th to the 1st lumbar segments the same divergence of the sclerotic stretch continues on either side of the median line until in these segments they occupy comma-shaped areas in the boundary region between the two posterior columns, reminding one of the topography of very early tabetic lesions. The commas lie directly across the path of the arcuate collaterals of the posterior root axones. Below the 11th dorsal segment the commas gradually diminish in size, until a trifle below the first lumbar segment they disappear altogether.

The histological structure of the sclerosis is quite that of ordinary mature sclerotic tissue in the white matter of the cord, viz., densely matted neuroglia hyperplasia with a wealth of larger and smaller vessels with thickened walls, many of which contain numerous investments of small round cells. Without reference to the clinical history, one may say positively that the sclerotic process has been of long standing. At the lowest estimate it has been in progress a year. Neither the upper, lower, nor inner median levels show any difference as to anatomical characteristics. It seems, therefore, impossible from consideration of the sclerosis alone to come to any conclusion as to where it began or where it was last in progress. Possibly the sclerosis in the cervical cord is oldest, as it seemed denser.

2. *Distribution of the Areas of Softening.*—In general the areas of softening involve the columns of Turck, the postero-lateral portions of the crossed pyramidal tracts, and the direct cerebellar tract and a zone of irregular width which wraps itself completely around the sclerotic lesion in the posterior columns. The upper vertical level of the softening reached the second cervical segment. The lowermost limit of the columns of softening lies in the fifth lumbar segment. The topography of the process of softening is in general so uniform that the description of one segment will suffice. The only additions we need make are as to certain variations in the lower lumbar segments, making it clear at first, however, that the softened areas in the antero-lateral and postero-lateral, and posterior columns are continuous in vertical extent. The softened tracts are columnar and not disseminated. In and below the first lumbar segment, all of the softened regions diminish in size. The columns of Turck, instead of being

entirely involved as in the preceding levels, have merely a small column on either side bordering the anterior fissure. The postero-lateral regions of softening are confined to the crossed pyramidal area; the posterior column softenings are two patches enveloping the lower attenuated extremities of the comma-shaped sclerotic areas described in the previous paragraphs. In the 4th and 5th lumbar segments the softened areas have disappeared entirely except two comma-shaped areas corresponding to the *bandelettes externes*. The structure of the softened areas is uniform. It consists of an open mesh-work composed of the strands and neuroglia septa strewn plentifully with Gluge's corpuscles. These softened areas are so porous that they stand out sharply in the Marchi and Weigert methods. The softening is obviously of very recent origin, within six weeks of the man's death at the minimum; and judging from the uniformity of the structure of the lesion it occurred at one stroke. All of the lesions of softening in this epoch of the history came on simultaneously.

3. *The Neuron Bodies and Spinal Roots*.—This may be dismissed in a few words. The roots, both ventral and dorsal, are normal. The neuron bodies in the main are also normal. Here and there, indeed, the ventral horn cells show eccentricity of position of the nucleus, but this has no significance. There is some central reduction of the Nissl plaques, but this is an exceptional circumstance undeserving of any prominence. Besides, there is the so-called pigmentary degeneration, which, seeing that it is found in individuals, especially of advanced age, who during life betray no symptoms, can hardly be taken into account in the discussion of our case. The blood-vessels, nerve fibres and neuron cell bodies of the oblongata, pons, and motor cortex are normal.

4. *Comparison of the Two Sets of Pathological Processes*.—

(a) *The Time Factor*.—With reference to the clinical phenomena we have here a case in which the spinal cord has been involved by two sets of lesions, the first occurring slowly and gradually, and the second terminal lesions occurring acutely and abruptly. While the first or sclerotic process was still in progress (or perhaps had become somewhat self-limited), a second set of lesions, entirely independent of the first, developed. These lesions took place a short time prior to the man's death, say some six weeks or so. The softening appeared as a complicating and terminal factor in

the course of the disease. The softening has an extensive distribution, but there is no doubt that all of the damage from this process occurred simultaneously, and must have made its appearance rather suddenly. This superimposition of one distinct process upon another takes the case out of the more common category of spinal disease, and makes the problem of the genesis of the two distinct sets of phenomena especially interesting. Unfortunately this point remains undetermined, as would naturally be expected from a study of a single case. Observing then the independence of the two sets of lesions from the obvious character of the histological structure alone, it is unnecessary to elaborate further points of distinction beyond the time factor in the questions of topography, course and vertical levels of limitations.

(b) *Comparison of the Clinical Phenomena and Anatomical Lesion.*—The clinical phenomena and anatomical lesions in general correspond very well. The man's history dates back two years. The wing-like process of the primary sclerosis of Goll's column extending into the Burdach territory correspond with the paræsthesia in the hands. The tremor of the hands is not so readily explained from the pathological lesions, because at this time there seems to have been nothing present in the cord except the sclerotic set of lesions, nor was there any interruption in the pyramidal fibres passing to the ventral horn cells in the lower cervical region. On January 1st, 1902, came the sudden and rather rapid onset of symptoms going in hand with the simultaneous development of the extensive columns of softening. Between the 1st of January and 11th of April, when the physical examination was made, which gives the most complete information of the clinical history, there was probably comparatively little change. In other words, had that examination of April 11th been made shortly after the 1st of January, it is quite likely that the condition would have been found materially the same.

While a consideration of lesions and symptoms have at least some features of simplicity in correspondence, the difficulties in the way of diagnosis during life were great. These two sets of pathological conditions of the cord must have been not inconsiderable. A consideration of the pathogenesis of the two independent pathological processes gives very little satisfaction relative to the causation. As regards the

origin of the posterior sclerosis, at least one factor can be excluded anatomically as well as clinically, and this is that the posterior sclerosis cannot very well be secondary to some transverse lesion in the cord, either of acute, subacute, or chronic formation. From an inspection of a single segment of the cervical segment one might infer the existence of a transverse lesion at lower levels. This particular cord, however, was divided into no less than 57 segments; each special segment was subdivided into three others; so any slowly-growing lesion interrupting at lower levels the fibres passing to the column of Goll in the cervical region, can be reasonably well excluded. The sclerosis then seems to be a primary one following pretty closely the system of fibres in the Goll's tract. The softening one would naturally consider to be due either to quantitative or qualitative changes in the vascular supply of the cord. Had the vascular changes, however, been of a qualitative nature, it seems hardly conceivable that the grey matter and neuron cell bodies could have escaped practically uniformly throughout the entire length and breadth of the cord. Quantitative fluctuations of sufficient extent to be antecedent to the considerable volume of softening would lead one to infer structural changes in the calibre or walls of the blood-vessels. Special attention was given to the condition of the blood-vessels. No thrombi, pathological changes in the vessel walls, aneurysmal dilatations, or even swelling of the endothelium and choking of the perivascular lymph spaces were found. The genesis of the softening is difficult to determine. That the softening was the forerunner of acute myelitis is out of the question, not only are there no signs of exudation and the attending phenomena of acute myelitis, but the length of time elapsing after the occurrence of the softening shows that it had remained at a standstill. It need not be specially emphasised how unlike this case is from those cases of systemic degeneration of the intraspinal sensory neurons associated with anæmias and structural disease of the blood-vessels.

## **CASE OF PNEUMOCOCCAL MENINGITIS AND SOME RECORDS OF THE VALUE OF THE CYTOLOGICAL EXAMINATION IN CASES OF MENINGITIS.**

By W. B. WARRINGTON, M.D., M.R.C.P.,

Physician David Lewis Northern Hospital, Demonstrator of Pathology  
in the University of Liverpool.

J. W., a boy *æt.* 8, admitted to the hospital on December 27th, 1903. We were told that he was apparently quite well on Christmas Day, but on the 26th he vomited several times and complained of headache; he appeared feverish and ill, and being worse on the following day was brought to the hospital. Previously to this illness he had been a healthy child, but six months ago sustained a severe fall which rendered him unconscious; for this condition he had also been treated at this hospital, and had had several convulsions. After a fortnight's treatment he had been discharged apparently quite well.

*State on admission* (27th Dec.).—The patient is a well-nourished child, lies on his back with the legs slightly flexed. He is in an irritable, semi-conscious condition, cries and moans, and complains of the pain in his head. Temperature 100°, pulse 108, respirations 36. Kernig's sign marked in both lower limbs. The next day he was quite comatose, though constantly crying. The head and neck were very slightly contracted, but forcibly rotated to the left; the facial muscles on that side were in a state of spasm, the angle of the mouth drawn up, the eyelid kept forcibly closed, and frontalis contracted into a unilateral frown. The left upper and lower limbs were the subject of jerky spasms. The right face and limbs were not observed to move, but were not in the state of resolution. Kernig's sign now absent; the knee-jerks and ankle-jerks were equal and moderately brisk. Right plantar flexor response. On the left side the great toe was strongly bent backwards. The pupils were moderately dilated, equal and contracted to light. No ocular paralyses, and the eyes constantly moved from side to side. The edges of both optic discs were blurred, and the veins large and distended. At 4 P.M., temperature = 101°, pulse 130, respirations 36. No sign of pulmonary complication was found. By lumbar puncture a markedly turbid fluid was obtained which rapidly

filled the collecting-tube; it contained a considerable amount of albumen. After centrifugalisation a very large number of cells were present, practically exclusively of the multinuclear type. The preparation also showed innumerable numbers of lanceolate diplococci. The cerebro-spinal fluid was inoculated on to agar agar and injected under the skin of a mouse. On the 29th, the following day, the patient obviously dying, the pupils were small and failed to react to light. A copious crop of small herpetic vesicles had developed during the night over the left angle of the mouth. The spasmodic condition had largely disappeared. The temperature rapidly shot up, reaching  $105^{\circ}$  before death, pulse and respirations also were markedly increased.

Death took place at 8 P.M. on the 4th day of the illness.

*Pathological report.*—The organism found in the C.S. fluid stained by the ordinary aniline dyes, and retained the stain by Gram's method. It grew in dew-drop colonies on agar, and was lethal to the mouse in thirty-six hours, from the blood of which animal the same lanceolate diplococcus was obtained. It was therefore Fränkel's pneumococcus.

On examination of the brain, the pia was injected of a bright red colour, the whole of the vertex, especially over the Rolandic areas, was the seat of patches of thick greenish pus. Similar patches were found in the ependyma covering the choroid plexuses and the outer and middle parts of the upper surface of the cerebellum. The veins of Galen were enormously distended, but there was not much excess of fluid in the ventricles. On the base of brain a thick patch of similar greenish pus was present in the interpeduncular space, extending along the Sylvian fissures and reaching forwards on to the frontal lobe, backwards slightly over the lateral lobes of the cerebellum.

The spinal cord was very slightly involved in its upper part.

The cavities of the ears, the frontal and ethmoidal sinuses were normal.

A small part of the base of the right lower lobe was found dark and airless; microscopical examination showed intense congestion and hæmorrhage, obliterating the alveoli, but no fibrinous exudation. The other viscera were throughout normal. There were no signs of tuberculosis.

*Remarks.*—This case appears worthy of mention as an example of a primary pneumococcal inflammation of the



meninges. No other viscus had been invaded. It illustrates the intensely acute nature of the illness, and the examination of the C.S. fluid demonstrated the etiology.

### I. POSTERIOR BASIC MENINGITIS.

G. A., æt. eight months, admitted September 21, 1901. A well-nourished child, lies on his back in a drowsy state, cries a good deal, vomits several times in the day. Marked retraction of head and neck, limbs flaccid. No Kernig's sign. Irregular fever. Rapid pulse and respiration. Examination of C.S. fluid, September 24: slightly cloudy, cells found are polynuclear forms in large numbers, some large and small lymphocytes. Diplococcus present, stained with carbol fuchsin and Löffler's methylene blue, loses stain with Gram's solution. Fluid non-pathogenic to a mouse and rabbit, and no growth obtained from inoculated fluid on agar, gelatin, or serum; this is possibly Still's diplococcus.

*October 1st.*—Child better; retraction of head diminished. C.S. fluid contains more lymphocytes than previously.

*November 7th.*—After several alternating remissions and aggravations of symptoms the patient is now much better.

C.S. fluid nearly clear, contains few lymphocytes. The patient was discharged three months after admission apparently recovered, but returned after some months with deafness.

### II. POSTERIOR BASIC MENINGITIS (under the care of Dr Bushby).

A. M., a girl æt. 16 months, admitted November 20th, 1901. Child has only been ill a few days. When first seen there was marked retraction of head and neck, almost opisthotonus; lower limbs rigidly flexed; Kernig's sign present. Optic disc red, but no neuritis. Abdomen rather distended. Temperature irregular, reaching in the evening 102°; pulse about 100°. Purulent discharge from nose and eyes.

C.S. fluid was turbid and contained great numbers of polynuclear forms of cells, small masses of pus were obtained. Examination on the 23rd showed increase of lymphocytes, but polynuclear varieties still predominated. The fluid was much

clearer. No growth was obtained by inoculation on to agar, glycerine agar or blood serum.

Death occurred on the 30th. The necropsy showed that the dura was markedly adherent to the skull along the middle line, great injection of vessels of the pia mater on the vertex with small amount of pus. At the base no pus visible, but distension of the arachnoid spaces at the posterior part of the brain. All the ventricles were markedly dilated and contained a semi-purulent fluid. The choroid plexuses resembled red currant jelly in appearance. The spinal cord was practically free. From the fluid beautiful examples of an intra-cellular diplococcus were obtained (Weichselbaum).

### III. POSTERIOR BASIC MENINGITIS (under the care of Dr Bushby).

Adult male: admitted with severe pain and marked retraction of head and neck, some opisthotonus. Kernig's sign present. Patient was in a semi-comatose condition.

C.S. fluid was very albuminous, contained a large number of cells almost exclusively of polynuclear type. Inoculation on to agar and serum gave no growth. At the later period the lymphocytes increased so that they were present in greater numbers than the polynuclear variety. The patient made apparently a perfect recovery.

### IV. VERTICAL MENINGITIS (under the care of Dr Bushby).

J. G., boy æt. 8 months, admitted October 7, 1901. Illness began about a week ago with vomiting and headache, three days later had bilateral convulsions. On admission the child lies in bed in a semi-comatose condition with all his limbs rigidly flexed. No retraction of head or neck. Optic discs normal. Fontanelles tense. The convulsions recurred and the child died two days after admission.

C.S. fluid withdrawn during life showed almost exclusively the polynuclear variety of cells. Inoculated on to media a staphylococcus was obtained. A mouse inoculated with the C.S. fluid died in 14 days and the staphylococcus was isolated from its blood.

The necropsy showed that the hemispheres were covered

with a thick purulent deposit, extending over almost all the cortex, but being especially marked anteriorly. There was also some pus at the base of the brain, especially in the interpeduncular space and extending backwards on to the cerebellum and along the spinal cord to the level of the fourth cervical nerve. Heart, lung and liver normal. The middle ear was normal. Spleen enlarged and a few large mesenteric glands.

#### V. TUBERCULAR MENINGITIS (under care of Dr Bushby).

F. D., child admitted with typical symptoms. The autopsy showed the characteristic lesions, confirmed by microscopical examination.

C.S. fluid showed lymphocytes in great predominance. No tubercle bacilli found.

#### VI. TUBERCULAR MENINGITIS.

J. S., boy æt. 9 years. Indefinite symptoms at first, later becoming characteristic of meningitis. Kernig's sign absent. A post-mortem showed extensive tubercular lesions.

C.S. fluid contained large number of lymphocytes of small and large variety, the small ones being more numerous. A moderate number of multinuclear forms. No tubercle bacilli found. In the early stages of illness, typhoid was thought of. This was negated by the examination of the C.S. fluid, and by a leucocytosis of 16,800 with a negative serum reaction.

#### VII. TUBERCULAR MENINGITIS.

J. H., boy æt. 6 years. Signs of meningitis. Kernig's sign present. Survived eight days after admission. Lumbar puncture performed three times. On each occasion a slightly turbid fluid withdrawn, which contained a preponderating number of multi-nuclear forms. Some lymphocytes present. A considerable amount of albumen was present, and a diplococcus which, however, could not be grown on agar. No tubercle bacilli found.

The necropsy showed most extensive dissemination of tubercles in the glands, lungs, spleen and kidney. In the right femur and tibia the epiphyses were the seat of purulent

tubercular disease. In the brain extensive meningitis along the sylvian arteries and circle of Willis. This extended on to the convexity, which, along the central convolutions, was covered with thick, semi-purulent, gelatinous lymph. A tubercular nodule was present in the vermis of the size of a hazelnut. The meningeal lesions may not impossibly be the result of a secondary infection engrafted on to the tubercular process. I have met with a diplococcus in other tubercular meningeal inflammations, but have never been able to isolate the organism (*vide* Case No. 8).

#### VIII. TUBERCULAR MENINGITIS.

W. B., boy *æt.* 20 months. Vomiting, diarrhoea, emaciation, and great retraction of head and neck. Necropsy showed typical tubercular lesions.

C.S. fluid contained lymphocytes in large numbers. No tubercle bacilli found, but a diplococcus present.

#### IX. TUBERCULAR MENINGITIS.

E. D., girl *æt.* 3. Formerly had "pneumonia" and right otorrhoea (probably tubercular) followed by complete facial palsy. Admitted with signs of meningitis. Kernig's sign absent. Necropsy showed typical tubercular disease of the meninges and other organs.

C.S. fluid showed great predominance of lymphocytes, but considerable number of multi-nuclear forms. Tubercle bacilli (acid-fast, attenuated, moniliform bacilli) present in fair numbers.

#### X. TUBERCULAR MENINGITIS.

G., boy *æt.* 8 months. Seen with Dr Buxton of Seacombe. Doubtful symptoms for ten days, slight vomiting previously. Lies curled up in bed and resists movement. Kernig's sign present. Tongue very foul. Bowels constipated. Pulse 72, regular. Temperature oscillating for the last week at about 99°.

The diagnosis was difficult. The C.S. fluid was clear but albuminous, and contained a large number of lymphocytes. No tubercle bacilli present. During the following days the symptoms increased in gravity, and death took place in about sixteen days after the onset.

## OBSERVATIONS.

The excellent paper with an account of the literature which has just appeared in this journal by Dr Macfie Campbell renders it superfluous for me to do more than allude to a few points and add one or two references to his list.

With his conclusions I entirely agree, and regard a positive cytological result from the examination of the cerebro-spinal fluid as the most valuable single objective symptom we are acquainted with.

In two of my cases Kernig's sign was absent, whereas the cytology of the fluid gave a positive answer.

Although in the great majority of cases it suffices to distinguish between the meningism of typhoid fever and a true meningitis, it is not absolutely to be relied upon, for pure lymphocytosis has been recorded in an early stage of typhoid by Mèry and Babonneix,<sup>1</sup> and by Variot, in which the after-history showed that presumably there was no gross meningeal lesion. I have also found a great preponderance of lymphocytes over the multinuclear forms in a case of hemiplegia in a child *æt.* four. This was probably syphilitic in origin, but the acutely ill condition of the patient might have suggested a tubercular meningitis.

With regard to the formula, in acute non-tubercular meningitis a leucocytosis is found; in the tubercular disease a lymphocytosis. This is undoubtedly a very constant rule. Pernet<sup>2</sup> analysed 86 cases at the Trousseau Hospital for the years 1901-1902: of these 53 were tubercular and followed the rule, but the multinuclear form predominated in four. Case 7 of my cases is another exception; it may be noted that here the lesions were very extensive both in the viscera and brain, and it is possible that some explanation may be established in these cases, as Macfie Campbell says it is not possible to exclude a secondary infection. When purulent patches of exudation are present it is very likely that multinuclear cells will appear in the C.S. fluid, irrespective of the nature of the primary lesion.

I have not found it necessary to follow so rigorously the technique laid down by Vidal. Careful centrifugalisation is

<sup>1</sup> Soc. de Pédiatrie, 18th February 1902.

<sup>2</sup> *Thèse de Paris*, 1903.

certainly necessary, and the deposit should then be removed by a capillary pipette. In fixing the films I find slowly applied heat satisfactory, and methylene blue or methyl green give quite good pictures.

The cells seem readily to undergo dissociation, and the granular are not often well defined by being stained with the special dyes, such as Ehrlich's or Jenners'.

The special micro-organisms cannot as yet be often easily found, especially in the case of the tubercle bacillus, and I was not surprised to read that Dr Trevelyan had not succeeded in doing this in the five cases he mentions in the Bradshaw Lecture. It was found in one of my six cases, and in a seventh by my colleague Dr Bushby, to whom I am greatly indebted for his kindness in permitting me to record the findings in those patients who were under his care.

## **A MORPHOLOGICAL CONTINUITY OF GERM-CELLS AS THE BASIS OF HEREDITY AND VARIATION.**

By J. BEARD, D.Sc.,

University Lecturer in Comparative Embryology, Edinburgh.

*(Continued from p. 34.)*

### **IV. THE REDUCTION OF CHROMOSOMES.**

A CURSORY survey of the immense literature of oogenesis and spermatogenesis would suffice to demonstrate that the question of the reduction of chromosomes cannot be fully discussed in these pages. It might appear to be foreign to the argument, but it is a phenomenon of universal occurrence in the Metaphytic as well as in the Metazoan life-cycle, and its nature is of fundamental import for problems of heredity and variation.

Owing to the duplication in the number of chromosomes which happens at every conjugation of egg and sperm, a reduction to half the number becomes necessary prior to a new conjugation, and hence either concomitantly with or prior to the formation of new gametes, eggs and sperms. This phenomenon of the reduction of chromosomes occurs, so it is generally

admitted, somewhere or other between the formation of oocytes and spermatocytes and the last division of these into cells, which become gametes (Figs. 8 and 9, p. 122). For a fuller discussion of the matter the reader may consult the memoir upon the "Determination of Sex." Among embryologists there are two views maintained. One of these is (1) that the reduction is effected by the disappearance of half of the chromosomes in the closing phases of the final division of the oogonia into oocytes and of the spermatogonia into spermatocytes (Boveri and others). The reduced number appears here, but how this disappearance of half the chromosomes is brought to pass neither Boveri nor any succeeding worker has yet been able to make out. Boveri remarks that the critical point at which observation is brought to a standstill is the nucleus of the oocyte and spermatocyte of the first generation, i.e. at the line o.c. and h.s.p.c. in Figs. 8 and 9 (p. 122). According to him, it is not the reduced number in the actual egg or polar bodies or sperm which requires explanation, but this diminished number in the oocytes and spermatocytes. (2) The second view (Weismann and his pupils and Rückert) is that the foregoing apparent reduction is not a real one, but that the actual reduction is effected in one or other of the two final divisions of the oocytes and spermatocytes. They maintain that these are preceded by a conjugation of chromosomes in pairs, and by one longitudinal splitting of each pair. The first division takes place along the line of the split, the second separates the conjugated chromosomes; therefore there would be no loss of chromosomes. On the other hand, Boveri and others hold, and in this they are supported by almost the whole of botanical opinion, that the two final divisions are preceded by *two* longitudinal splittings of the reduced number of chromosomes. Under this view there is thus an elimination of chromosomes.

This latter point is a very important one, for, if it be true, and to my mind the real evidences seem to speak for it, the products of any oocyte or spermatocyte, usually four in number, will be identical gametes, and not such containing different combinations of characters, as Weismann maintains. Under the simple views adopted in the present writing, the egg or sperm contains within itself one complete set of the characters requisite to form or unfold an individual of the species, like one complete

pack of cards of a certain colour, red, blue, or green. Moreover, for reasons already entered into elsewhere and repeated in another part of this writing, it is concluded that some important phenomenon takes place in the final division of the oogonia, for example, into oocytes, and that whatever be the causal connection of this (i.e. the reduction) and the determination of sex, the two are linked together and inseparable. If the reduction be the outward and visible manifestation of the determination of sex, it is easy to understand why it should happen in, for example, the passage from oogonia into oocytes, for undoubtedly the final phenomena of the determination of sex take place here.

If the view advocated by Weismann, Rückert and others be correct, there can be no connection between the two. This would then be an inexplicable circumstance, and the whole thing with its (under their views) associated phenomena would reveal such remarkable conditions as almost to be beyond comprehension.<sup>1</sup>

And it appears to be certain that in the mammalia, where only one polar body has been described in all known cases, there cannot be two divisions of the oocytes. Again, Meves has quite recently published in preliminary form some very interesting observations upon the spermatogenesis of a wasp, *Vespa germanica*, and the bee, *Apis*. At the moment the finer details are wanting, but Meves records that in both cases there are the usual two final divisions of the spermatocyte into spermatids. The results of these closely resemble the diagram of oogenesis (Fig. 8). In both cases the first division yields a normal cell and a sort of "polar body" containing no nuclear substance, while the second in *Vespa* results in two normal spermatids, which become two sperms, and in *Apis* it furnishes one normal spermatid and a "polar body" containing nuclear substance, but none the less rudimentary in nature. Thus, in these instances,

<sup>1</sup> The view of the reduction adopted in the present writing is that established by the researches of Boveri, Brauer, O. Hertwig, Meves, Strasburger and others, and it was formerly also advocated by Farmer and Moore. In a recent publication (*Proc. Roy. Soc.*, London, vol. lxxii. p. 104-108, 1908) the two latter investigators have arrived at other conclusions, which bear some resemblance to those advanced by Haecker and Rückert. Boveri's view has the enormous advantage that under it the phenomena of the determination of sex and those of variation, etc., admit of simple and natural explanation. And, moreover, it is supported by the work of some very fine observers.



notwithstanding the twofold division of each spermatocyte, in *Vespa* but two sperms arise, in *Apis* but one. The work (10) will probably turn out to be of great importance in the light it must throw upon the reduction, and it may quite conceivably finally render the views of Weismann as to the reduction untenable, if they be not so already.

These latter seem to me to lead into what in research is always carefully to be avoided, a cul-de-sac. There are already more than sufficient cul-de-sacs in embryology without making new ones or helping to maintain any now existing, and in these pages Boveri's conclusions as to the reduction will be adopted; that is to say, it will be taken as established, that the reduction of chromosomes is effected—not by cell-division—in the closing phases of the final division of, for example, the oogonia into oocytes. Prior to this reduction the nuclei of germ-cells may, and often do, exhibit the duplicated condition and, of course, always the doubled number of chromosomes, and the interpretation to be placed on the matter is that it represents a doubling of all the characters or qualities requisite to form an individual. These, therefore, are twofold, like two packs of cards, red and blue respectively, in the primary germ-cells, and in all the cells, until oocytes and spermatocytes are formed, that is, until the final phenomena of the determination of sex take place. As will be seen presently, this conclusion is of far-reaching import for the problems of heredity and variation.

*Postscript.*—In a recent publication, received during the correction of the final proof-sheets, Boveri has considerably modified his earlier views, vide “*Ergebnisse über die Konstitution der chromatischen Substanz des Zellkerns*,” Jena, 1904.

## V. THE DETERMINATION OF SEX.

Until 1902, all current views as to the determination of sex were based upon the assumption that in the Metazoa there existed but *two* sorts of gametes, the egg and the sperm. And so sure were embryologists of the truth of this, *that they had never attempted to establish it in fact in any one case!* As long ago as 1854 Bernhard Schultze, from the consideration of the facts relating to double monsters and identical twins, had concluded that *two* sorts of eggs, male and female respectively in

destination, must exist. If this were true, and it certainly is so, underlying the phenomena of sex there must be at least *three sorts of gametes*, the sperm, the male egg, and the female egg. A fourth kind of spermatozoon, non-functional, had long been known in *Paludina*, a fresh water dioecious snail. It was discovered in 1836 by the celebrated zoologist, C. T. von Siebold. Since then second forms of sperms, or the forerunners of such, all non-functional, have been recorded in many animals from various divisions of the animal kingdom, and this number is constantly being added to. So that, indeed, it is known for far more instances, that two forms of sperms or their forerunners occur, than that only one sperm is developed. And of dioecious animals as yet closely studied there are perhaps only two, *Ascaris* and *Salamandra*, where a second can with any certainty be said to be absent. In these instances the very numerous degenerate spermatogonia and spermatocytes sufficiently account for its non-occurrence.

The memoir, of which a brief abstract is given in the following, starts by confirming Schultze's conclusion, given above. Soon after it was published, two others appeared, by M. von Lenhossék (11) and Oskar Schultze (12) respectively. These embryologists also confirm the conclusion, that underlying the phenomena there are two kinds of eggs; the latter writer, from the results of an extensive series of experiments upon mice, demonstrates that no external conditions, such as hunger, rich food or poor, age or other factors, can influence the determination. The three memoirs, Lenhossék's, Schultze's, and the writer's, treat of the question independently and from different points of view. But they are in complete agreement so far as the fundamental facts are concerned, that underlying the phenomena there are three kinds of functional gametes, and that the actual determination of sex lies with the female Metazoan organism, as she alone differentiates among her germ-cells two forms of functional gametes, the male egg and the female egg. Neither von Lenhossék nor Schultze enter at all into the discussion of the general questions, which are of extreme morphological and embryological interest and importance, of the nature of hermaphroditism, the phenomena of parthenogenesis, or the universal occurrence of two forms of sperms, or their direct forerunners, in male Metazoa. The former writer, in a short examination of the facts relating

to the parthenogenesis of the bee, arrives at conclusions as to their meaning very similar to mine.

Sex in its origin in the Metazoa would appear to have been bound up with the constant differentiation of gametes of *four* categories. These were such that two of them, the female and the male eggs, were formed within a sterilised Metazoan person, the female, while the development of the remaining two, the two kinds of spermatozoa, was allotted to a similar, but not identical, person, the male. The gametes of the female, that is, the two forms of eggs, possessed functions different from those of the two kinds of spermatozoa of the male, and for this reason alone a sexual difference between the male and the female was bound to follow.

Of the twofold gametes of the male it is to all appearance rare at the present time to find the full and complete differentiation of both in any given case, but this is known to happen in *Paludina vivipara* (von Siebold, M. von Brunn, F. Meves), *Pygaera bucephala* (F. Meves), and a few other instances. In others one form of male gamete undergoes more or less complete suppression in the course of the spermatogenesis, thus in *Cicada tibicen* (E. V. Wilcox), *Bufo calamita* (von La Valette St George), *Hyla* (von La Valette St George), *Bombinator* (Ivar Broman), etc., the degree of degeneration varying in different cases. Though never of functional value, unless it take the place of the ordinary form of sperm, the second kind of spermatozoon is probably always represented by something in every Metazoan spermatogenesis, its development must at least be initiated, but it may be arrested anywhere in the history of the spermatogonia or spermatocytes. It has been described by more than a dozen observers in certain members of the following divisions of the animal kingdom: Mollusca, Nemertea, Rotifera, Arthropoda, Amphibia, Aves, and Mammalia, including man himself (in the latter by von La Valette St George, K. von Bardeleben and von Widersperg). In animals belonging to the aforesaid groups its occurrence has been put on record in about 40 species. But all observers agree, that it is never functional.

Holmgren has recorded two kinds of spermatozoa in one of the Coleoptera, and this suggests the inquiry, whether the second form of sperm may not in fact sometimes or often be functional, as well as the usual sperm. The order of Coleopterous insects

is very large, embracing not fewer than 70,000 species. The Insecta form an immense group, not less than 250,000 species being known. Probably, in a thousand years, the spermatogenesis of not a tithe of either the Insecta or its order of Coleoptera will have been worked out. This reflection bears significantly upon the question of the relativity of human knowledge. Naturally, we cannot wait another thousand years before drawing our conclusions: we can only say, that in our experience a second form is never functional, and that, though it be very often differentiated, there is no great likelihood of its ever being found to be of functional value alongside the usual form of sperm and in addition to this.

The exceedingly interesting and important recent work of Meves, referred to above, affords additional evidence of the probable truth of this. Meves has studied the spermatogenesis of *Paludina* in much greater detail than any preceding observer. He records, that in the spermatogenesis of *Paludina* there is no reduction of chromosomes in the spermatocytes of the worm-like sperm. A most curious irregular reduction takes place in the first of the two ensuing divisions, and the final result is, that each spermatid, resulting from the second mitosis, contains but one chromosome. The normal number of chromosomes in *Paludina* is, according to Meves, 14, the reduced number 7. Here, therefore, instead of 7 chromosomes each spermatozoon contains but the equivalent of one. In *Pygaera*, according to the same observer, in the formation of the non-functional form of spermatozoon chromatic material, in other words chromosomes, find no place, "in *Pygaera* the second form of spermatozoon is completely destitute of nuclear portion, that is, it is headless" (Meves). Notwithstanding Meves' cautiously expressed opinion to the contrary, there would appear to be no escape from the conclusion that, even the complete differentiation of the second form of sperm in *Paludina* and *Pygaera* being accompanied by phenomena, only diagnostic as degenerative, it can be of no functional import whatsoever.

When, therefore, it is stated that evidences of degeneration are always to be found in the development of a second form of sperm, and that these and other evidences, i.e. actual experience, always and invariably point to its non-functional nature at the present time, this conclusion is in accordance with the strictest

canons of scientific investigation. The opposite view would be not only contrary to all experience but, apparently, incapable of proof. "But," it may be asked, "may not some one or other of the 250,000 species of insects possess two forms of functional spermatozoa?" This is exceedingly unlikely, and it would not fit in with the homogeneity of the reproductive processes of the Metazoa as they now exist. One might as well hope to meet with cases in which the polar bodies of oogenesis were normal functional gametes or eggs. Neither contingency is, of course, impossible, only highly improbable, for what has been in the past may, so far as our very limited knowledge and intelligence extend, happen again in the future. Neither occurrence would merge into the miraculous, as is the case with the supposed conversion of males into hermaphrodites. We may neither limit Nature's powers, nor seek to make her perform miracles.

It is the task of the functional spermatozoon to bring about the effects due to the union of two germ-cells, of two individualities or sets of characters. Since it is the egg which develops, and not the sperm, the burden of providing for the continuance of the race falls upon the female Metazoon, or, rather, upon the germ-cells, of which it is the host. To carry out this duty the differentiation of twofold gametes, the male eggs and the female eggs, is needful. The germ-cells of the female thus make provision not only for a new batch of female eggs, but also for one of male eggs. On the other hand, the germ-cells of the male only furnish one form of *functional gamete*. *The determination of sex for the next generation thus lies with the germ-cells of the female Metazoan organism.*

Under the generally accepted but erroneous view of the existence of only two forms of gametes, eggs and "ordinary" spermatozoa, in the Metazoa but one form of individual can arise. Ordinary hermaphroditism illustrates the truth of this, for here all the individuals arising from the union of such germ-cells are alike. For the constant production of two forms of individuals *three* categories of gametes at least are needed.

In all dioecious Metazoa *three kinds of functional gametes* are constantly required and differentiated; of these *two* arise in the female, *one* in the male. (Figs. 8 and 9.)

Dioecious and hermaphrodite Metazoa may be defined as

animal forms, in which in every sexual individual either two categories of gametes, or the forerunners of such are constantly differentiated. Of these gametes never more than three sorts are functional (dioecious forms), and there may be but two such (some hermaphrodites).

The actual determination of sex is initiated at the division of the primary germ-cells into secondary ones: it is completed at the formation of the oocytes and spermatocytes, and its mani-

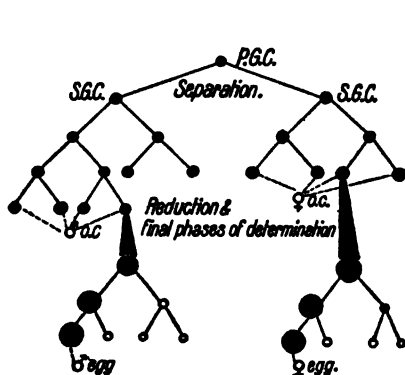


Fig. 8.—Oogenesis and determination of sex in *Raja batia*.

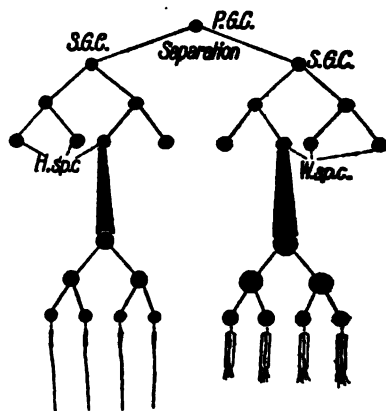


Fig. 9.—Probable course of spermatogenesis in *Paludina* (after the statements of F. Meves).

festation is accomplished by the numerical reduction of the chromosomes in these.

While to all appearance the determination of sex would be effected in plants at the formation of the spore-mother-cells<sup>1</sup>, it does not come about in their equivalents in animals, the primary germ-cells; for if one of these undergo independent development alongside the embryo, the result is the bringing forth of identical twins. All known cases of such are of the same sex, and from this it would follow, that all the primary germ-cells of a given case are alike in sexual potentialities as in other respects. All the evidences go to prove that the determination of sex does not take place later than the formation of the oocytes and sperma-

<sup>1</sup> A view apparently also held by Strasburger (*Biol. Centralbl.*, Bd. 20, 1900, p. 769).

toocytes. The history of the two sorts of eggs of *Hydatina senta*, *Dinophilus gyrotilatus*, *Phyllozera*, and *Raja batis*, and that of the twofold spermatozoa of *Paludina* and *Pygaera* suffice to demonstrate the truth of this.

The faculty of becoming hermaphrodite is confined to the female. The male can only produce spermatozoa, often of two kinds, but only one form of functional gamete. The female produces two kinds of functional eggs, from one of which, the male egg, she is able on occasion by anticipation to form spermatozoa. (Fig 10). In all described cases of hermaphrodite (male) individuals among vertebrates the forerunners of a second form of sperm (spermatogonia or spermatocytes) have been erroneously taken to be "eggs."

*Hermaphroditism* is associated with the partial or complete suppression of one form of gamete, the male egg; *Parthenogenesis*, on the other hand, entails the occasional, or the cyclical, arrestment of one or other of the two gametes of the female. If it become acyclical (Weismann), with the consequent disappearance of the males, with these there vanish the male eggs, which produce them, and the spermatozoa. In such instances the only form of gamete left is the female egg, which, as is well known, undergoes an isogamous union with a rudimentary sister, the polar body.

Of very great importance for many questions is the recognition that any particular form of gamete may undergo suppression at any period of the life-history; thus, in some instances of the rare production of male persons their occasional reappearance is undoubtedly due to the omission to suppress one or more of the forerunners of male eggs. Similarly, the rarity or the apparent absence of a second form of spermatozoon in some instances is readily explicable.

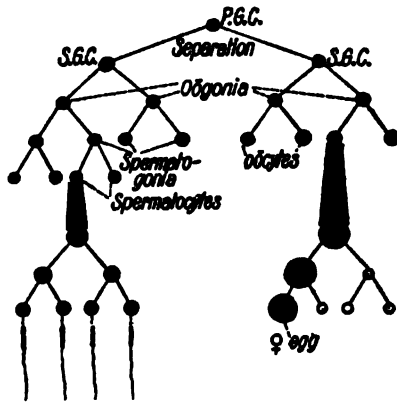


Fig. 10.—Supposed course of oogenesis and spermatogenesis in hermaphroditism.

Of the problems of sex three aspects stand contrasted: these are its origin, its regulation, and its determination. Of its origin no absolutely certain knowledge is possible: probably it arose from an original isogamy passing into a heterogamy of fourfold gametes, thence passing ultimately to the present actual heterogamy of three forms of functional ones, with frequently a fourth but non-functional one.

The experiments of Yung, Born, Maupas, Mrs Treat and others are often regarded as dealing with the origin of sex during development, or with its determination. In fact these observers have endeavoured to regulate it; for they have all started from the erroneous assumption, that the animals used in their experimental researches were either of no predestined sex or hermaphrodites. This error completely vitiates their results, which only prove what percentage of either sex will survive under given, usually utterly abnormal, conditions. The recent results of Cuénot and Oskar Schultze completely disprove the possibility of altering or modifying sex during development.

The regulation of sex in nature is, as Hensen and Düsing thought, self-adjusting. Like the determination it lies with the female organisms. The male has as little influence upon its regulation as upon its determination. The total of the females of a race occupy the same position towards the regulation of sex, as the individual female does towards its determination. How the self-regulation is brought into operation is described more fully in the original memoir. The basis of the principle is simply preponderance of males in the earlier, and of females in the later offspring. An increment in the race is effected by increasing the number of offspring, and with these the number of females. The effect of this begins to manifest itself in the third generation.

All interference with, or alteration of, the determination of a sex, is apparently absolutely beyond human power. To hope ever to influence or modify its manifestations would be not less futile and vain than to imagine it possible for *man* to breathe the breath of life into inanimate matter.

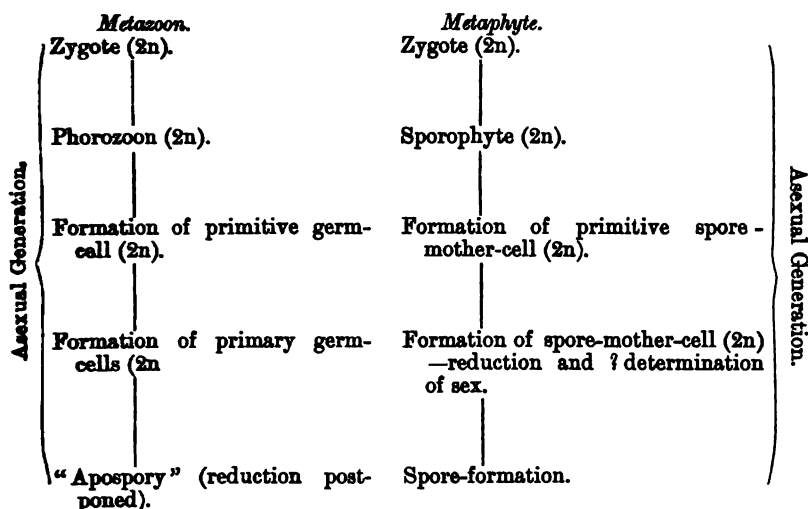
For the workings of Nature in sex merge in her revelations of Life itself.



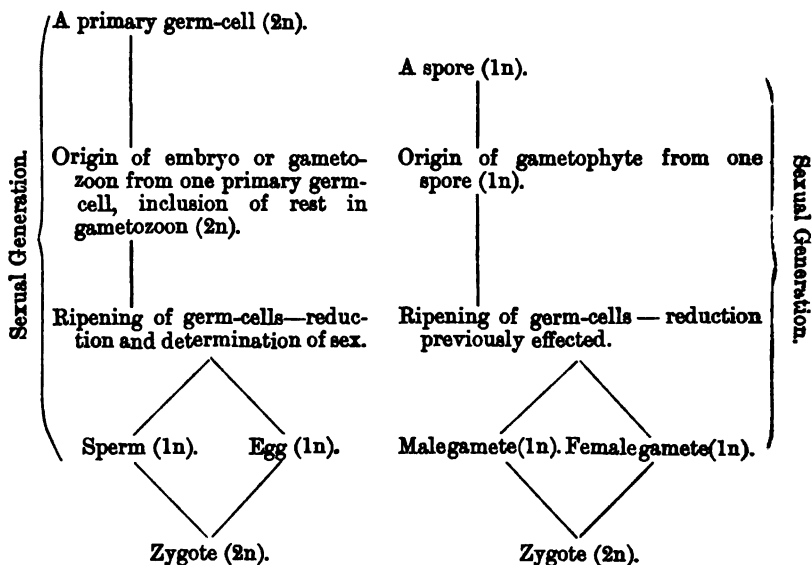
# VI. COMPARISON OF THE LIFE-CYCLES OF THE HIGHER ANIMALS AND PLANTS.

The theory of an antithetic alternation of generations as the basis of Metazoan development postulates something resembling the formation of spore-mother-cells in plants. It is clear that the reduction of chromosomes has been deferred to a later portion of the life-cycle in the Metazoa as compared with plants; and this fact was insisted upon by J. A. Murray and the writer in 1895. At that time we compared the two modes of development together in tabular form, and we postulated the formation of the embryo in animals upon the asexual generation or phorozoon from a spore-mother-cell. Certain facts supporting this view were cited, including E. B. Wilson's "teloblasts"<sup>1</sup> of the earthworm, which must be derivable from a single cell. Finally, the missing "spore-mother-cells" have appeared in the primary germ-cells.

## REVISED COMPARISON OF METAZOAN AND METAPHYTIC LIFE-CYCLES.



<sup>1</sup> These are a few large cells, comparable to the two cells *m* of Fig. 7. They proliferate chains or bands of cells destined to form various parts of the worm within the larva. Excellent figures of them will be found in Wilson's "The Cell," 2nd ed., 1900, p. 374, Fig. 175.



In the above table “ $n$ ” equals the number of chromosomes prior to the duplication, “ $2n$ ” at conjugation, that is, fertilisation.

In 1895 the writer was not sufficiently sanguine to believe it possible that at present the embryo or sexual generation would be found to arise from such a spore-mother-cell in any instance. Only its formation at some time in the past from a spore-mother-cell was spoken of, because the facts of development at that time known seemed to point to its origin from at least a few cells. And, moreover, everything then seemed to go to prove the production of the “sexual organs,” the germ-cells, by the embryo itself.<sup>1</sup> Such was the belief of almost every embryologist, and there appeared little or no reason for doubting its correctness. The effect of these two factors was to bar further progress in that direction, at any rate for a time. In face of the apparent facts, I confess that it was impossible to

<sup>1</sup> That is to say, from the germinal epithelium of Waldeyer, and as so derived there was a somatic origin, not a morphological continuity. Between 1895 and 1900, nothing occurred to alter embryological opinion. At the close of 1900, when the words in the text were originally written, the first account of researches upon the germ-cells of *Raja* was published. These facts throw light on the question of the priority of the discovery of a morphological continuity of germ-cells, which by Waldeyer is now assigned to Boveri and Weismann, whose published researches really demonstrate no continuity of germ-cells.

foresee how the formation of the spore-mother-cell was effected. Moreover, there was not the slightest suspicion in Murray's mind or in mine, that the germ-cells had anything to do with the matter.

It is possibly a humiliating confession to make, but it is true, that I was never able to conceive how Nature could carry out this formation of a spore-mother-cell, until my researches had revealed how she actually accomplished it. No one could have been more astonished than the writer at the revelation. Never for a moment had it been imagined that the germ-cells themselves would play the part they actually do in the life-drama of an antithetic alternation of generations. Only when the first portion of the work was practically complete and ready for publication, was it seen that the missing link in the alternation had at length been discovered in the primary germ-cells and in the epoch of their formation. I hardly feel called upon to prove that the primary germ-cells do represent spore-mother-cells; it is so obvious. Were each of them to undergo a reduction with the subsequent production of four "spores," and were then each animal spore to develop into an organism, we should have the exact equivalent of the gametophyte of one of the higher plants. Instead thereof, they remain together, and only one becomes sterilised to form a sexual individual or gametozoon (to contain the remainder). Their remaining together, and the continued and progressive amplification of the gametozoon in course of ages, have naturally deferred their ripenings, sex-determinations, and reductions to later and later periods. It is obvious that this could easily be effected by starving them, but this may not have been Nature's method of delaying their ripenings.

In the higher plants it is the spores, whose name is legion; while the "sexual cells," eggs and sperms, are few and far between. In animals the "sexual cells" exhibit the reverse condition, corresponding in their multitude to the spores of plants; while, as we at length know, the spore-mother-cells—there are no spores in the Metazoa—are not very numerous,<sup>1</sup> being represented in

<sup>1</sup> Seeing that the original primary germ-cells, whatever be their number, at later periods undergo many divisions, and that apparently this number can perhaps with the course of time and on occasion be still further added to, it might seem to be a matter of no consequence whether the number of primary germ-cells was, for instance, 2 or 512. The meaning of the observed differences in the number is, to some extent, bound up with the origin of the mode of the development.

some cases by but one cell in addition to that which forms the sexual generation.

Why this difference? In the embryo-sac of *Pinus*, which is the gametophyte, there are only four germ-cells. In the corresponding structure in flowering plants there are perhaps three, or at most six or eight; while, as is well known, the male gametophyte of a flowering plant is represented by one or two vegetative cells and one or two germ-cells. No Metazoan sexual generation has so small and scant an endowment as these; while such an animal may contain and harbour a number of germ-cells thousands of times greater. The difference is doubtless partially due to the different procedure adopted at the formation of the primary germ-cells or spore-mother-cells. The plan carried out in animals has been such as to favour the ever greater and greater amplification of the sexual generation. In plants, as elsewhere already insisted, the reverse is the case. Here the asexual generation has undergone increased amplification without ever being able to attain to any very high degree of histological differentiation. The sexual generation of plants is at the best a miserable failure from the morphological point of view, and this must be set down to the factors indicated, and still more to others to be yet described. The higher one ascends, the smaller it becomes, until, in the highest flowering plants, it has almost reached the vanishing point, without, however, being able to disappear entirely.

In animals it is the phorozoon or asexual generation which makes the bravest show in the lower Metazoa; but even here it is usually overshadowed in degree of morphological differentiation by the embryo or sexual generation. In the higher forms it becomes reduced; but, like the rudimentary sexual generation of the higher plants, *it cannot vanish, for it also has its assigned task in the reproductive round.* The sexual generation or gametozoon, thanks to the importance of the precious cargo of germ-cells which it carries, has received the kindly attentions of Nature, with consequent higher and higher evolution. From a variety of causes the phorozoon, on the other hand, tends to simplification the higher one ascends. At the best, its organisation is simple, but this simplicity even leans to meagreness in the vertebrata as they now exist, and in the mammalian chorion or

trophoblast the phorozoön has reached the lowest depths of degeneracy, for its structure is almost devoid of histological differentiation.

## VII. THE METAZOAN LIFE-CYCLE AND ALTERNATION OF GENERATIONS.

Modern embryological doctrine is at least consistent in holding to the three tenets of somatic origin of germ-cells, direct development, and epigenesis. If the germ-cells be somatic in origin, direct development is *a priori* possible. If the hen produce from her body new eggs, it is at all events feasible that any one such egg should develop directly into a new hen, and if this happen, there would appear to be little or nothing to hinder the development from taking on an epigenetic character.<sup>1</sup>

As, of course, every tyro in embryology knows, the doctrine of epigenesis was established in 1759 by Caspar Friedrich Wolff, who "demonstrated,"<sup>2</sup> in the instance of the chick, how from an apparently undifferentiated mass, what we should now term the cleavage products, part was gradually added to part, very much in the same way that one may any day witness the building of a house from the heap of material, bricks, boards, beams, windows, and what not, lying upon the ground beside the site. Study the developmental history of an earthworm or leech, the whole development of a skate or mammal, and the possibility of epigenesis may appear in another light. But, whether epigenesis be possible or not, nothing is more certain than that by no modern observer has it ever been proved to be the mode of origin of an embryo underlying the development.

<sup>1</sup> Weismann has already more than once (in the preface to the "Germ-plasm" and in the "Vorträge," vol. i. p. 385 *et seq.*) declared against the possibility of an epigenetic development. I find myself in complete accord with him in this conclusion, and my researches may claim to be the first embryological ones to support his attitude in this important matter. My reasons are briefly given in the present section. But, on the other hand, Weismann has never questioned the possibility of either direct development or somatic origin of germ-cells, "in some cases at any rate," for the true Metazoa. The writer finds it difficult to understand how he can still accept these two doctrines, which would appear to render an epigenetic development not merely possible, but likely. If ALL the products of the egg-cleavage be merely bricks for the embryonic body, how are they to form this without the builder, epigenesis?

<sup>2</sup> It perhaps need hardly be stated, that to-day the demonstration of 1759 would require to take on a very different character!

Incidentally, be it added, the foregoing passage and the doctrine of epigenesis itself show how great is the importance of "the embryo" in the embryological mind, and how little weight is really laid upon other phenomena of the life-cycle prior to its appearance.

The foundation-stones of epigenesis are direct development and a somatic origin of germ-cells. If these be removed, the whole structure of epigenesis will crumble away. Of the three tenets previously mentioned, possibly to none do embryologists cling with greater tenacity than to direct development. In the Metazoa it is looked upon as the rule: indirect development is, indeed, recognised under the term "metamorphosis," though, doubtless, no advocate of direct development has ever formed, or been able to formulate, any clear, logical conception, free from metaphysics and "Naturphilosophie," of what he understands by "metamorphosis." The term "metamorphosis" is not originally a scientific one at all! It belongs to the realm of the supernatural, and it has been imported into science—from mythology!—to account for facts, which at best it scarcely succeeds in explaining away. To take an instance, the *trochophore* larva of an annelid worm is said to become "metamorphosed" into the annelid. Were this ever really to happen, the phenomenon would be exactly comparable to that which in mythology occurred when Jupiter turned himself into a bull. A construction of this nature is not scientific, and no explanation of this kind, given of a developmental process—embryology teems with such accounts of phenomena—will bear the analysis of the microtome and microscope. A juggler may succeed in every attempt to deceive his audience into the sure belief that he has converted an old hat into a live rabbit, but the method and details of the "metamorphosis" will not permit of a closer inspection. Surely, notwithstanding the circumstance that writers of text-books may state the metamorphosis of the *trochophore* into the annelid as a proved fact, there is to-day no practical embryologist prepared to maintain it? Neither as a whole, nor as to the parts of its body, neither as an organism, nor as a series of organs, does the larva become changed into the worm.

In 1886 the late Professor N. Kleinenberg set up the doctrine of "development by substitution of organs" (13). His conclusions were based on this very instance of the developmental

facts relating to the *trochophore* and the annelid worm. He had found that the *trochophore* possessed a nervous system, quite distinct from that of the future worm, not homologous with this, and such an one that the future central nervous system of the worm did not arise from it, but, on the contrary, the larval one degenerated. From these and other facts, at the close of a fine and laborious investigation, he came to the conclusion that for any given organ, for example, the nervous system, in the course of the ancestral history the original structure had become insufficient, and had been replaced by a new organ arising from a different source. The old organ appeared in the development, because the stimulus it produced was needed for the production of the replacing organ. Applying this principle generally to all the organs, he concluded finally, that in development there was a substitution of organs, corresponding to that which had occurred in the history of the race. This is, of course, really an attempt to place the recapitulation theory on a scientific basis. In 1896 the writer suggested a modification of the idea in proposing to recognise, not so much development by substitution of *organs*, as development by substitution of *organisms*. Kleinenberg's doctrine is false, because for every organ, A, B, C, D, E, etc., in the worm, there is not a corresponding organ a, b, c, d, e, etc., in the larva. If capital letters be taken to denote the organs of the worm or sexual generation, and small letters those of the larva, the latter may be made up of a, b, c, d, e, f, g, while the former will be composed of A, B, C, D, E, F, G, H, I, J, K, L, M, N, O, P. Moreover, A does not replace a, B oust b, and C substitute itself for c, etc., at various periods of the development, corresponding to the periods at which this may be presumed (without evidence) to have happened in the ancestral history, *but all the facts go to show that at a certain epoch, the critical period, A + B + C + D + E + F + G + H + I + J + K + L + M + N + O + P begin to substitute themselves for, i.e. to suppress a + b + c + d + e + f + g, the latter all then beginning to degenerate. These are plain, simple, elementary facts of development*, not to be found in any text-book extant, but which can easily be verified in a worm, or a fish, or a mammal. For the annelid they were first really described without the true interpretation by Kleinenberg, for the fish and mammal by the writer in his published researches. But clearly a + b + c + d + e + f + g together make up the parts of the larva,

A + B + C + D + E + F + G + H + I + J + K + L + M + N + O + P those of the worm. Therefore, the conclusion may be drawn that the worm as a whole, as an organism, replaces another organism, the larva. Or, in other words, the development is one of substitution of organisms. But it is something more than this. The organs of the larva are not homologous with any like-named organs of the worm, and neither organism as a whole is homologous with the other, and so this substitution of organisms is in reality an instance of antithetic alternation of generations, the worm being the sexual organism or generation which succeeds and replaces the asexual generation or larva. Although the facts—if none be ignored or distorted—only admit of this construction, few embryologists are supporters of it. Although direct development is no explanation whatsoever of the facts, none the less, it is still the creed, nay, the superstition, of the majority of embryological devotees. The idea of direct development is probably as old as the intellect of man himself. This “scientific fact” was naturally one of the first to arrest his attention, and even to-day can it not be verified within the space of three weeks in any poultry-yard? *And yet it is a supposition incapable of proof and impossibly true!*

Underlying the dogma of direct development is a somatic origin of germ-cells. This is not really avoided by the assumption, made by Waldeyer and von Lenhossék, that the fertilised egg at its first cleavage separates into two portions, one destined to form the germ-cells, the other “the embryo.” And as little are the real facts of the case met by Nussbaum’s (14) aphoristic statement, which has a look of some mythological account of the creation about it, “es theilt sich das gefurchte Ei in die somatischen und in die Geschlechtszellen.” Should these observers ever attempt to verify their doctrines in the actual facts of development, even in those already recorded by the writer, they would at once recognise how untenable their positions were. No instance is known, none is likely to be, in which the course of events is as suggested by them.

But there is, fortunately for the seeker after fact, one division of the Metazoa to which, while the phenomena of the life-cycle therein observed are rather complicated, no one—at all events nowadays—seeks to apply the doctrine of a metamorphosis, viz., the Hydrozoa or Hydromedusae. Here, as Weismann demon-



strated in 1883, the germ-cells arise in an asexual stock, the hydroid polype or colony of such; and thence they migrate into the medusa or its equivalent. This is not termed a metamorphosis, but an alternation of generations. In such an animal as the skate the phenomena are exactly comparable. Within a living structure, the blastoderm, there arise germ-cells, which upon the appearance of an embryo wander into it. Is it at all remarkable that, as the blastoderm and its appendages never become "metamorphosed" into the skate, one should also describe this as an alternation of generations?

By looking at the phenomena in this light one immense advantage is gained, in that thereby it becomes possible to compare together, so as to show their essential identity, the phenomena in the life-cycles of a hydrozoon, a worm, a mollusc, an insect, a fish, a mammal, and a man. And to these must be added the higher plants or Metaphyta. In other words, *it reveals the unity of organic nature!* What other view of development possesses this enormous advantage? What upholder of direct development, or of epigenesis, can produce a diagram of the life-cycle of a Metazoon from egg to egg, corresponding to and including all the known facts of the development, and confirming to the hilt his view of the process? There is no ring of uncertainty in the answer! Apart from that drawn up by the writer, there exists no other complete diagram of the life-cycle. For no Metazoon has it been established beyond cavil and doubt that the development is direct. If there be any such thing as direct development, where under it do the germ-cells arise, and what is their cell-lineage from the fertilised egg? It is easy to ask these questions, but the difficulties in the way of answering them are so great that they never will, or can, receive convincing replies in the sense of a direct development.

On the other hand, it may confidently and emphatically be stated that under the conception of an antithetic alternation of generations all the phenomena observed in the development of any Metazoon, from a hydroid polype to man himself, are capable of ready and simple explanation. On this view of matters it is clear why in the Nemertean life-cycle the worm should oust the *Pilidium* larva, why the starfish should substitute itself for the *Bipinnaria*, why two distinct and separate nervous systems should appear in a certain definite order in any development,

why these should differ so markedly in morphological features as do that of the *trochophore* and that of the annelid, or as do the transient nervous apparatus of the skate-development and that of the sexual generation, the skate itself. It explains why there may be two distinct nervous systems, and not three, four, or more. The doctrine of direct development is impotent in face of these and similar facts. But possibly there is a sort of explanation given by the recapitulation-theory, under which every animal recapitulates more or less completely and perfectly its ancestry in the course of its individual development. There are various difficulties in this theory; in fact, it bristles with them. Like many other current beliefs, *it was originally never founded in observation at all*, and it is still largely advocated in spite of all observation. Were there a basis of truth in it, it ought to be easy for any embryological upholder of the doctrine to give an outline of, say, the ancestral history of the vertebrata, or of the mammalia. For the latter class, since the days of the late Professor W. K. Parker, no one has ever attempted to explain the facts in the light of this theory, while for the former the name of the theories supposed to be founded in recapitulation is legion; moreover, each excludes the others.

But suppose recapitulation were really the true explanation of the facts of development, and not as it actually is, but the illusion of the human imagination, under it there would be daily happening in every development an alternation of generations, beside which that actually demanded by Nature for her workings would be mere child's play! She requires, according to the view of Metazoan development which my researches have forced me to adopt, two organisms in every life-history.—an asexual one, upon which the germ-cells can arise from one or more "apical cells," and following upon this a sexual organism to contain and nourish these germ-cells for a certain span of time. This appears reasonable enough upon the surface. On the other hand, it may be asked, would it be reasonable or necessary for her, in order to bring any and every developmental history to stand, to call up from the dim shadows of the past a spectral and fantastic procession of ancestral forms?

The crucial points upon which the doctrine of development must stand or fall, are without question the nature of so-called larval forms and the source and time of origin of the germ-cells

To explain larval forms as ancestral is simply to beg the question, for no living being knows, or can know, that they are such things. Echinoderms flourished as long ago as the Lower Devonian shales of Lynton, in North Devon. This was millions and millions of years ago, how many millions it is difficult to estimate. In reality it does not matter for the question under discussion whether the old fossil-bearing rocks of Lynton be 5 million or 500 million years old. Even were they but 5 million years old, a brief span of time as geological history goes, who shall say, and whence shall he obtain the information, that then and earlier modern Echinoderm larvæ existed as organisms, with sexual organs (!!!), ancestral forms, of which the *Pluteus*, *Bipinnaria*, etc., are modern pictures? There are no larvæ preserved in either the older or newer formations. The thing from its nature is an assumption incapable of proof, and, though a never-ending multiplication of causes, it offers an entirely inadequate explanation of the facts.

Even more serious for direct development is the question of the epoch of appearance and the source of origin of the germ-cells. A morphological continuity denies their somatic origin. And if they be not somatic, with the actual facts of development as they are, with the pre-embryonic appearance of the germ-cells, a direct development is inconceivable, is as impossible as epigenesis.

On the one hand, there are the three doctrines of somatic origin of germ-cells, direct development, and epigenesis; on the other, a like number, morphological continuity of germ-cells, antithetic alternation of generations, and evolution, or unfolding.<sup>1</sup> All the known facts of development support the latter trilogy. It is out of question to discuss, on the present occasion, all the bearings of these three doctrines, or even to foresee these. Of necessity they are connected together; each is an essential part of the theory of development. Outside embryology and zoology their bearings upon scientific problems will undoubtedly be great, but here it may be instructive to point to one or two little things, which they help to explain in zoology and embryology. The position of the sponges, and their mode of development,

<sup>1</sup> Differs from the older views in this way. The evolution of Bonnet, Haller, and others, was an evolution with preformation, that of Weismann is an evolution with predestination.

have been never-ending themes of discussion with zoologists, while the evolution of the mammalian trophoblast has not been less fruitful of speculation. For brevity the facts may be placed in tabular form:—

## LIFE-CYCLES.

Name.	Asexual generation.	Sexual generation.
Nemertine	<i>Pilidium</i>	nemertean worm
Sponge	sponge-larva <i>equals</i> adult	absent
Sea-urchin	<i>Pluteus</i>	sea-urchin
Hydra	<i>Hydra</i>	absent
Hydroid polype ( <i>Campanularia</i> )	hydroid colony	<i>medusa</i>
<i>Raja batis</i>	blastoderm, transient nervous system, etc.	skate
Mammal (e.g. man or rabbit)	trophoblast (chorion)	mammal

As the table shows, the sponges and *Hydra*, like the corals (Anthozoa), are forms in which, as in *Fucus* among plants, the sexual generation is not represented by anything unless by eggs and sperms, while in the mammal the asexual generation, which makes so brave a show in the Hydrozoa, etc., has become reduced to the almost structureless trophoblast.

## VIII. THE NATURE AND TRACK OF HEREDITY.

In other chapters the essential similarity—the equivalence—of all the primary germ-cells, whether their number be two, sixteen, one hundred and twenty-eight, or anything else, has been insisted upon. The point is one of the utmost importance, and, therefore, it may be well to indicate briefly once more the grounds for the conclusion. All the primary germ-cells have the same ancestry from the primitive germ-cell. One of them goes to form an embryo; and there is nothing to show that this one differs in any respect from its sister-cells.<sup>1</sup> If two primary germ-

<sup>1</sup> In *Strongylus*, Spemann has commented upon the equivalence of what he terms the primitive germ-cell and the primitive mesoderm-cell (embryonic-cell or somatoblast); indeed, he speaks of them as "Geschwisterkind" or cousins (*Zool. Jahrb. Morph. Abteil.*, vol. viii. p. 313). His primitive germ-cell is, however, a primary germ-cell; and the true primitive germ-cell is that from which the two cells compared together took their birth.

cells undergo independent development on a blastoderm, the result is and must be the production of identical twins. The dermoid cysts or embryomata of Wilms are, as this able investigator has established, rudimentary embryos.<sup>1</sup> These abnormal embryos must have taken their origin from persistent primary germ-cells; and the development of an embryoma is embryologically the abnormal formation of a twin, identical with the "embryo" or individual containing it.

The likeness of all the primary germ-cells is certain, or almost so; absolutely nothing suggests unlikeness among them. This essential identity or equivalence of all the primary germ-cells is immensely important from the point of view of heredity. This will be quite obvious. It is it, and it alone, which permits of the handing-down of the characters of one generation to future generations. It is the very basis of heredity. The formation of like primary germ-cells, and their essential similarity or equivalence, show how, in sexual reproduction, the "offspring" resemble their "parents," while differing from them. The likeness in the primary germ-cells leads to likeness in the offspring; and along with this, unlikeness is bound to come in. For the primary germ-cells themselves give rise to secondary germ-cells, which have apparently lost their powers of independent development. It is these, and these only, as a rule, which normally are present in the finished embryo. In the Vertebrata, at least, they and their progeny never undergo independent development; but it is their destiny to go through the process of reduction of chromosomes, with the ensuing formation of gametes, eggs or sperms. Here, as is of course now generally recognised, unlikeness enters. The egg or sperm traces its long "ancestry" to one of a certain set of primary germ-cells, of which one also gave rise to the "embryo" or form whose "offspring," according to social and commonly accepted ideas, the egg or sperm itself was. This latter unites with another sperm or egg, the offspring of a different individual, which in its turn, with its reproductive elements, traces a similar origin and ancestry from another set of primary germ-cells. With the union the new cycle begins.

It is thus that the formation of primary germ-cells underlies

<sup>1</sup> For a fuller account of the facts and conclusions in this direction, see the short paper on "The Embryology of Tumours" in list of literature, and for the complete paper a forthcoming part (1904) of the *Journ. of Anatomy and Physiology*.

the fundamental facts of heredity, and explains these. And it is thus, without their knowing it, that the formation of the primary germ-cells at a certain epoch of the development, prior to the production of an embryo, is the real basis of Weismann's finds in heredity, and, to a still greater degree, of those associated with the name of Francis Galton. The latter has been led by his studies and researches on inheritance to what is known as "Galton's law." According to this law, "the two parents between them contribute on the average one-half of each inherited faculty, each of them contributing one-quarter of it, The four grandparents contribute between them one-quarter, or each of them one-sixteenth, and so on; the sum of the series  $\frac{1}{2} + \frac{1}{4} + \frac{1}{8} + \frac{1}{16}$ , etc., being equal to 1, as it should be. It is a property of this infinite series that each term is equal to the sum of all those that follow, thus:  $\frac{1}{2} = \frac{1}{4} + \frac{1}{8} + \frac{1}{16}$ , etc.;  $\frac{1}{4} = \frac{1}{8} + \frac{1}{16}$ , etc., and so on. The prepotencies and subpotencies of particular ancestors in any given pedigree are eliminated by a law that deals only with average contributions, and the varying prepotencies of sex in respect to different qualities are also presumably eliminated."

It may be well to state here, that in the later chapters dealing with variation some new light will be thrown upon fundamental questions of heredity. These novel points cannot be anticipated at the present juncture, and merely for purposes of argument Galton's law will be assumed to be correct. Under this assumption and under that of a constant environment, its embryological basis is furnished by the formation, etc., of the primary germ-cells. The germ-cells in any embryo, possessing from their mode of formation like qualities, and having these and the like ancestry with that which formed the embryo, these qualities are necessarily halved at the following reduction and determination of sex. At the close of this halving the "parental" qualities can only, *under Galton's law*, be represented by at most one-half, or one-quarter for each "parent," and so on for each preceding generation; for in these also primary germ-cells of like characters were formed, of which one gave rise to an embryo in every case. The line of ancestry is, of course, from and through these germ-cells, and never from the embryo or sexual generation of any preceding generation.

But as the germ-cells associated with any given embryo are

all of like characters among themselves (including that from which the embryo arises), on the production of eggs and sperms, and the subsequent union of these with other sexual products, the result is the same as if the line of ancestry had been through the embryo, so far, at any rate, as the ancestral characters are concerned. In the same way, and because between offspring and grandparent there are two sets of germ-cells (in addition to those still immature in the offspring) and two reductions, the grandparental portions taken together can only be half of the parental portions taken together, that is to say,  $\frac{1}{2}$ , and so on through any number of generations. It will be needless to carry out the examination further.

In addition to a constant environment, Galton's law postulates complete blending in reproduction and the absence of inbreeding. W. K. Brooks has already drawn attention to the latter point. He has insisted that Galton's theory demands absence of relationship among all the ancestors. He then goes on to show, that in the case of three people living on a small island, their known ancestry goes back seven to eight generations. The maximum number of distinct ancestors for all these three persons together should be 1146, according to Brooks. Of these 452 are recorded, but these are not 452 distinct persons, being, in fact, only 149 (15).

But Galton's law may be regarded from still another aspect, and this arises from the following embryological facts. In its origin the reduction of chromosomes was possibly merely an undoing of the previous union, and even now it is never the halving of one unit, but of *two* such. Therefore it is not a reversion to half cells, or half entities or individualities, but to *whole ones*. From this it follows, that at fertilisation we have to deal with the union of two individualities of two complete lines of ancestry.<sup>1</sup> The union of these is continued in the primary germ-cells, as evidenced by their duplicated nuclei, until the initiation at least of the ensuing determination of sex, and the united lines are broken up into separate complete lines, not necessarily identical like strings of many coloured beads with the original lines, at the ensuing reduction and sex determination.

<sup>1</sup> The reader may be reminded that in this portion of the text it is being assumed that the reduction is an indiscriminate halving of all the characters. That it is not really such is demonstrated in another chapter.

All along the line, from the fertilised egg to that primary germ-cell which unfolds as an embryo, this duplication is evident, and of course, it must at first be in this cell too. As I have recognised in embryological lectures during the past four years, there must be a competition between the two components of the duplicated nucleus, when development begins. This will be such, that of the total nuclear constituents, which together make up the inherited characters of the two "lines," one-half must be suppressed, or more probably remain latent.<sup>1</sup> If these characters be symbolised by the letters of the alphabet, in such a way that the first half of these represent the characters of the one line, the second half those of the other, in the development of the embryo only the half of this total can be made use of. Where one letter drops out, its place will be occupied by the corresponding letter of the other half of the alphabet.

The conception of "heredity," as the origin of the word indicates, is taken from material mundane affairs of mankind. In development there is no inheritance or handing-on of anything at all from a Metazoan individual of one generation to its "offspring," a Metazoan individual of the next generation. Embryologically, we have no ancestors, no parents (except unicellular ones), and no offspring. In this way it is seen, that a new conception of the nature of what we term "Heredity" is needed. And it must be clear that the "theory of heredity" outlined in preceding pages, and which will receive still further and, I venture to believe, important reinforcements in the chapters upon variation, has little or nothing in common with previous ones. Underlying it is something more than a mere morphological continuity of

<sup>1</sup> How easy in embryological discussions it is to use similar words with very different meanings is shown by the following passage of Haecker's, referring to this duplication and its import. "Eine ähnliche Concurrenz kommt vielleicht auch in den Bildern aus den Gonadanlagen von *Diaptomus* zum Ausdruck, und würde für das Verständniss mancher Vererbungserscheinungen (Dominiren des einen Elters) von Bedeutung sein" (*Anat. Anz.*, vol. xx. p. 451). Since this was written by Haecker in 1901, his complete memoir has appeared. The writer anticipated seeing in this a long discussion of the point, instead thereof the latter work (p. 376-378) contains little more, and nothing more of importance, about it than is given in the few lines quoted above. Haecker looks upon the thing as merely indicating which of the two nuclear halves shall dominate in the development, the paternal or the maternal. It may be added, that his memoir (p. 348 *et seq.*) contains an account of the instances in which the nuclear duplication, etc., has yet been recorded or described. (Haecker, V., "Ueber das Schicksal der elterlichen und grosselterlichen Kernanteile," in *Jen. Z. Naturwiss.*, vol. xxxvii. p. 295-400, 4 plates, 1902.)



germ-cells. From its nature it might be termed "the Understudy Theory of Heredity." Given in a certain life-history the period of formation of the primary germ-cells. Of these let there be for simplicity but two, AB and BA. On one of these falls the lot of developing into an embryo: to which of the two this happens is not of consequence for the argument. In all its essential characters the remaining primary germ-cell (whose immediate destiny it is to become the founder of the "sexual products" of the said embryo), is the exact counterpart of the developing one. So much so is this the case that, if both form embryos, these are identical twins.

In the ancestry neither of the primary germ-cells, AB and BA, had ever been a Metazoon; neither they nor their ancestors had ever formed parts of a Metazoan body. But their ancestry is continuous with a long line of germ-cells, and at regular intervals these were exactly like certain sister-cells, which did develop and form Metazoan individuals. Although the cell AB does not itself give rise to an embryo, in the meantime it retains for itself, and also for all its immediate progeny, the properties of BA, those characters which, were it or any of its progeny to develop, would make it or them identical twins with BA, the other cell which did develop. *This is the greatest wonder in embryology!*

In the drama of heredity there are always understudies, which for a certain essential period are endowed with all the identical properties of that germ-cell from which the player arises. These understudies, the primary germ-cells, are never employed upon the stage as such—except in the instances of identical twins, triplets, etc.—but some of them in new guises and after new conjugations are the immediate ancestors of those which become the acting characters in new scenes of the cyclical drama of Life.

*(To be continued.)*

#### ERRATUM.

Page 34, line 3. Instead of "*interrupted*," read "*uninterrupted*."

#### REFERENCES.

10. Meves, F. "Ueber 'Richtungskörper-bildung' im Hoden von Hymenopteren," in *Anat. Anz.*, vol. xxiv. p. 29-32, 8 figures, 1903.

11. Lenhossék, M. von. "Das Problem der geschlechtsbestimmenden Ursachen," Jena, 1903, p. 1-99.

12. Schultze, Oscar. "Zur Frage von den geschlechtsbildenden Ursachen," *Arch. Micros. Anat.*, vol. lxiii. p. 197-257, 1903.
13. Kleinenberg, N. "Die Entstehung des Annelids aus der Larve von *Lopodorhynchus*," in *Zeitschr. wiss. Zool.*, vol. xlv. p. 1-227, 1886. *Loc. cit.* p. 212-224.
14. Nussbaum, M. "Die Vererbung erworbener Eigenschaften," in *SB. Niederrhein. Gesell.*, Bonn, 1903, *loc. cit.* p. 2 (of separate copy).
15. Brooks, W. K. "The Foundations of Zoology," p. 143-145.

## Abstracts

### ANATOMY.

#### "THE LIMBUS POSTORBITALIS" IN THE EGYPTIAN BRAIN.

(38) G. ELLIOT SMITH, *Anat. Anz.*, Bd. xxiv., H. 5 and 6, 1903, pp. 139-141.

G. RETZIUS, Giacomini, Eberstaller and E. A. Spitzka have already called attention to the fact that part of the orbital region of the cerebral hemisphere may slip back over the orbito-sphenoid into the middle cranial fossa; but these writers regard it as a rare condition, since Eberstaller found it only three times in 200 (? German) brains, and Retzius only eight times (in its well-developed form) in 100 Swedish brains. In 64 hemispheres of Egyptians, Soudanese, and Balkan people, I found it in 39 cases. [Since my note was written I have examined 200 brains, and find that the limbus is present on one side (at least) in two-thirds of the specimens. In 3 per cent. of the cases in which it occurs it is equally well developed in both hemispheres; in 5 per cent. it is larger on the right side; whereas in 92 per cent. it is distinctly bigger, or (more often) alone present on the left hemisphere. The fact that it occurs equally in three such dissimilar groups of people as the Soudanese, Egyptian, and Balkan races—I have seen large limbi in a scaphocephalic Soudanese with a cephalic index of 58, and in a hyperbrachycephalic Armenian with an index of 91—makes it hard to believe that it is so rare in other people as Eberstaller's statistics would have us believe. This is a point worth noting in European brains, because the confirmation of Eberstaller's results would constitute this a most important distinctive feature of the European as contrasted with the various Asiatic and African types of brain.]

AUTHOR'S ABSTRACT.

**ON THE INTRA-CELLULAR NETWORK OF GOLGI OF THE  
(39) NERVOUS ELEMENTS OF THE SPINAL CORD IN THE  
ADULT SUPERIOR VERTEBRATE.** SOUKHANOFF, *Journ.  
Ment. Pathol.*, Vol. v., No. 1, 1903.

THE author has succeeded in demonstrating the intra-cellular network of Golgi in the nerve cells of the spinal cord of adult rabbits by the following method. The animal was killed by chloroform, the cord extracted and split longitudinally down one side. Transverse incisions were then made at a distance of 0.5 cm. from each other and the small pieces thus obtained used for the research.

They were placed in Veratti's fluid for twenty-five days, thence into a mixture consisting of three parts of 5 per cent. bichromate of potassium and one part of 5 per cent. copper sulphate. In this they remained two and a half days, and thence were placed in 2 per cent. silver nitrate for from one to three days. After being passed rapidly through celloidin, they were cut, cleared in guiacol, then turpentine, and mounted in balsam without a cover-glass.

The intra-cellular network was found in both large and small cells, and the author verifies Golgi's observation that it is arranged around the nucleus, a free space being left between it and the cell periphery. Processes are given off into the dendrites of the nerve cells of the spinal cord and cerebral cortex, but in the spinal ganglia the network appears like a closed sac without any processes.

Finally, the network has nothing in common with the fibrils of Apathy and Bethe, nor with the endo-cellular reticulum of Donaggio. It may, however, have something in common with the intra-cellular spaces described by Holmgren, Studnicka, Bochenek, and Donaggio, and may be analogous to what Nelis calls "état spirémateux."

DAVID ORR.

**THE COMPOSITION OF THE POSTERIOR COLUMNS OF THE  
(40) SPINAL CORD.** KURT GOLDSTEIN, *Monatsschr. f. Psychiat. und  
Neurolog.*, Bd. 14, H. 6, Dec. 1903.

THIS paper is entitled an anatomico-critical study. The anatomical part of it consists of a detailed account of the histological examination of (1) a case of compression of the cauda equina, and (2) a case of tabes dorsalis.

(1) A tumour had compressed the roots of the cauda equina without direct injury to the cord, and the latter had been examined by the Marchi and Weigert-Pal methods, with special reference to the changes in the posterior columns. The posterior roots were found degenerated as high as and including the second

lumbar on both sides, but the lower sacral were not so seriously injured as the higher sacral and lumbar roots.

In the posterior columns of the lower sacral region there was general diffuse degeneration with, however, an area, corresponding to the dorso-medial bundle of Obersteiner, or sacral triangle of Gombault and Philippe, which contained relatively few degenerated fibres (Marchi). The same median zone was more degenerated in the middle sacral region, and in the upper sacral region assumed a shape resembling the oval area of Flechsig, but containing again relatively less degeneration. In the 5th lumbar segment the median zone was hardly distinguishable from the surrounding degenerated field, and above this level it disappeared. The ventral zone of the posterior columns was degenerated from the coccygeal region upwards, the degenerated fibres being more numerous than in the median zone, but less than in the remainder of the posterior columns as high as the 2nd lumbar segment. In the 5th lumbar segment there appeared a small area (postero-external field), lying close to the median side of the entering root, which contained very little degeneration, and this zone was distinguishable as high as the 2nd lumbar segment.

In the 5th lumbar segment, too, there was less degeneration on the dorsal periphery of the cord close to the median septum; this relatively free zone (bandelette) was seen also in the higher lumbar segments, but better by the Weigert-Pal than by the Marchi method, and was soon lost in the lower dorsal region, where it lay more laterally.

(2) A case of tabes dorsalis examined by the Weigert-Pal and carmine methods of staining. The coccygeal and two lower sacral posterior roots were only partially degenerated; the 3rd sacral to 2nd lumbar roots were markedly degenerated; the 3rd lumbar to the 9th dorsal roots were slightly degenerated; the 8th, 7th, and 6th dorsal roots were less degenerated; the 3rd dorsal to the 8th cervical roots were almost completely degenerated, but above this level the roots were practically intact.

Beside the general diffuse sclerosis associated with the entry of sclerosed roots, sections from the lower sacral region showed slight degeneration in the median zone; those from the upper sacral region marked degeneration in the same area. In the lumbar cord the median zone (oval field) was relatively intact. The ventral field showed a few degenerated fibres in the lower sacral region, but more in the upper; in the lumbar cord the degeneration was more marked in the medial than in the lateral parts of the ventral field. A patch of intact fibres appeared at the level of the 4th lumbar segment in the region of the intermedial septum, and presented the appearance of a modified comma tract, which could be traced more or less distinctly as high as the upper dorsal region.

The number of intact fibres in the comma and in Hoche's peripheral tract at any particular level appeared to bear some relation to the degree of sclerosis in the entering roots of the segments immediately above that level. Hoche's peripheral bandelette, owing to its relative preservation, could be traced from the 2nd lumbar segment, where it was in connection with the median triangle as high as the 5th dorsal segment, becoming more lateral in position and effecting a junction with the thickened dorsal extremity of the comma tract. The postero-external zone referred to in the first case also showed marked degeneration in the segments lying below the most degenerated posterior roots.

Both the above cases were carefully worked out and described in more detail than space allows here. The author then proceeds to the critical part of his paper, which may be said to include a discussion of the literature on the subject, and the conclusions he draws from it and from the findings in his own two cases. In reviewing the data—histological, embryological, experimental, and pathological—which have been accepted by the majority of authors as proving, or nearly proving, the endogenous origin of the well-known descending tracts in the posterior columns—the comma, Hoche's bandelette, the oval area, and the sacral triangle—Dr Goldstein justly points out that they are by no means conclusive, that their evidence is largely negative and often open to a different interpretation. He proceeds, however, to maintain that his cases prove without any doubt the exogenous origin of practically all the fibres, not only in the tracts just named, but in the cornu-commissural zone, and in the postero-external zone as well. According to his view the endogenous fibres occupy no special tracts, but are scattered diffusely through the posterior columns, are few in number, and only of short length. Whatever is the truth as regards the origin of the descending tracts in the posterior columns, we have no hesitation in saying that Dr Goldstein has not *proved* their exogenous source, and that the interpretation which he puts on the appearances found in his two cases is often open to criticism. No attempt has been made to reconcile the fact that in transverse lesions of the cord continuous degeneration has been traced downwards in the posterior column tracts for more than twenty segments, with the fact that in all pure root lesions, hitherto published, the degenerated descending fibres disappear a few segments below the lesion. Dr Goldstein's views would for many reasons be acceptable and helpful, but they lack substantial support in the present state of our knowledge.

An incomplete bibliography is appended, but the writer of the article does not appear to be acquainted with the important papers of Bruce and Muir on the subject.

E. FARQUHAR BUZZARD.

**NEW METHOD OF STAINING NEURO-FIBRILLÆ.** S. R. CAJAL,  
(41) *Compt. Rend. Soc. Biol.*, Dec. 18, 1903.

THE author proposes the following technique, which he considers superior to that of Bethe and of Scinarro.

1. Immersion of small pieces of nervous tissue in a solution of silver nitrate of  $1\frac{1}{2}$  to 6 per cent. (according to results desired), for a period of from two to ten days, at a temperature of  $35^{\circ}$  to  $40^{\circ}$  C.

2. Wash in distilled water for two minutes.

3. Transfer to following solution:—

Pyrogallic acid	1 gramme.
Distilled water	100 c.c.
Formol (commercial)	5-10 c.c.

Pieces are left in the solution for twenty-four hours.

4. Wash in distilled water for two minutes.

5. Harden in alcohol 90 per cent., then 95 per cent. or absolute.

6. Embed in collodin, celloidin or paraffin.

7. Thin sections; mount in Canada balsam or dammar resin.

The metallic precipitate obtained shows a remarkable affinity for the very finest nerve fibrils, which come out red-brown or even black against the transparent yellow background of the rest of the tissue.

A solution of silver nitrate of  $1\frac{1}{2}$  per cent. is recommended as the most useful; it shows well the intranuclear network of Mann and of Lénhossek. Ten days or less suffice for impregnation. The disadvantages of this weaker solution are that pericellular arborisations are only faintly coloured, and the outer portions of the tissue may shrivel a little. Even with stronger solutions and shorter immersion this latter disadvantage can scarcely be avoided.

S. A. KINNIEB WILSON.

## PHYSIOLOGY.

**THE IMPOSSIBILITY OF THE NEURONE THEORY.** C. DURANTE,  
(42) *Rev. Neurolog.*, Nov. 30, 1903, p. 1089.

THE simplicity of the neurone theory as propounded by Waldeyer brought it into almost universal favour, but the grounds on which it rested are not facts but the interpretation of observations, which later investigations have in part disproved, in part necessitated different interpretation.

Durante's aim in this paper is to demonstrate that the ganglion cell and its so-called processes are different organs to some extent independent of one another, and that the latter is a pluricellular structure. Further, to verify the individual

independence of the constituent neuroblasts in various pathological states, and to support the physiological individuality and activity of the constituent parts in nervous transmission.

His arguments may be grouped into:—

- (1) Embryological; for recent investigations have shown that the peripheral nerves are formed by the migration into the mesoderm of neuroblasts, which then elongate and fuse by their ends to form a continuous chain, each member of which differentiates individually, forming neurofibrils, which fuse with those of the neighbouring units.
- (2) Histological.—(a) The continuity of the axis cylinder is disputed by several recent workers, who rather attribute to each interannular segment the characters of nervous cells with partially differentiated protoplasm.
- (b) Apáthy, Bethe and others have shown that the neurofibrils of neighbouring neurones are continuous and anastomose with one another, besides forming extracellular nets.
- (c) The disproportion in bulk between a long axis cylinder and the cell to which it is attached, makes improbable the origin of the one from the other.
- (3) Pathological.—Wallerian degeneration, the strongest support of the neurone theory, is not absolutely exact.
- (a) The degree of degeneration in the peripheral end of an injured nerve is not identical in all segments, and several observations have been published in which the axis cylinder had not undergone degeneration though the cell was partially or wholly destroyed.
- (b) It is not a true degeneration, but a cellular regression of the constituent neuroblasts consequent on the cessation of functional activity.
- (c) Retrograde degeneration of the central stump is a fully confirmed fact.
- (d) The change is frequently not limited to the neurone affected.
- (e) Then, too, there are numerous facts in clinical pathology inexplicable or incompatible with the neurone theory—the unequal affection of the various segments of a nerve in neuritis, and the relative integrity of the distal end of a nerve which has been more or less destroyed by a neuroma or other tumour nearer the cells.
- (4) Regeneration.—(a) Several authors have seen autogenous regeneration of the peripheral end of a sectioned nerve.
- (b) Others have described the independent formation of neurofibrils in the neuroblastic (internodal) segments of a

regenerating nerve as occurs in embryological development.

- (5) Physiological.—(a) That nerves are not merely inert conductors of excitations, but impulses, during their passage along them, are increased and reinforced. This tends to show that the segmentary neuroblasts take an active part in the transmission of impulses, a fact which would be inexplicable under the neurone theory.
- (b) The relative slowness of the propagation of nerve currents is explained by regarding the latter, not as analogous to true currents, but as a transmission of excitation from neuroblast to neuroblast, each of which is active in the process.

GORDON HOLMES.

#### ON THE QUESTION OF THE RELATIONSHIP BETWEEN THE

- (43) **PATHS OF INTER-CELLULAR CONDUCTION AND THE PERIPHERY OF THE NERVE CELL.** DONAGGIO, *Riv. Speriment. di Freniatria*, f. 4, 1903, p. 825.

THE author admits that the peripheral reticulum in the nerve cell formerly described by him corresponds to that of Golgi, and is connected with fibrils in the surrounding tissue. These fibrils have been shown to be neuroglial—a view which is upheld by Bethe and Held, so that Donaggio is inclined to believe that his network is certainly not nervous but probably neuroglial.

Between the meshes of this peripheral reticulum there is a second system of very fine fibres forming a fine meshwork. The fibrils of this second reticulum are not so well stained as those of the first, and are said by Held to constitute a nerve current receiving apparatus, as, according to this author, nerve fibrils have been observed to end in it. Donaggio has never seen neurofibrils terminate in his second reticular apparatus, and draws attention to the deceptive appearances produced by Held's technique. Donaggio has employed a selective staining process for the reticula, which never stains the nerve fibrils. He is of opinion that the peripheral apparatus of the nerve cell is not conductive in function, and advances the hypothesis with reserve, that the first described reticulum may be nutritive in function.

DAVID ORR.

#### THE PARALYSIS OF INVOLUNTARY MUSCLE, WITH SPECIAL

- (44) **REFERENCE TO THE OCCURRENCE OF PARADOXICAL CONTRACTION.** H. K. ANDERSON, *Journ. of Physiol.*, Vol. xxx., 1903, p. 290.

It was shown by Budge in 1855 that after section of the left cervical sympathetic nerve and of the branches above the right



superior cervical ganglion in a young rabbit, the right pupil was smaller than the left immediately after the operation, but that twenty-four hours later the two pupils were equal, and forty-eight hours afterwards the right pupil was larger than the left. This greater dilatation on the more paralysed side has been termed by Langendorff "paradoxical pupil-dilatation." The condition has been fully investigated by Anderson, especially in young animals, and the term has been extended by him to include other cases in which after section of nerves having a tonic motor effect a greater contraction than normal is obtained. Under certain conditions the muscles paralysed by removal of a superior cervical ganglion show signs of greater tone than the corresponding muscles of the opposite side, both when the control sympathetic is uninjured and when it has been divided. This effect has been observed one day after the lesion, and more than a year afterwards. It is readily evoked by excitement, dyspnoea, and anæsthesia, and is noticeable after death. The paradoxical effect persists even when the post-ganglionic branches of the ganglion have been allowed to regenerate after previous section has set up the condition. If the animal which has had a superior cervical ganglion removed be cautiously examined without occasioning any excitement, the pupil on the side of the injury shows greater paralysis, and there is no paradoxical effect; but on occasioning any excitement, however slight, the paradoxical dilatation occurs. It is produced most readily in the kitten, and is less easily obtained as the animal grows older. Diminution or increase of light, and the application of atropine or eserine, do not prevent the paradoxical effect.

The views regarding the cause of paradoxical dilatation are discussed, and the author believes with Lewandowsky that the paradoxical effect is due to an increased excitability of the contractile tissues on the side without a ganglion; there is an actual increase of tone in the dilatator on the side without a ganglion. There is evidence against the views: (1) that the sphincter is weakened on the side without a ganglion (Budge, Tuwin, and Levinsohn); (2) that there is permanent contracture of the vessels of the iris, or dilatation on this side (Surminsky, Langendorff); and (3) that on the side without the ganglion the dilatator is inhibited (Kowalewsky).

The accentuation of the paradoxical effects seen under the influence of anæsthetics does not occur until anæsthesia causes dyspnoea.

A similar but weaker paradoxical effect may be produced by division of the cervical sympathetic alone, which shows that the ganglion cannot inhibit the automatic excitability of the contractile tissues with which it is left in connection.

Anderson finds no evidence in favour of Weymouth Reid's view that the inhibition of the dilatator is effected through the 3rd nerve simultaneously with contraction of the sphincter.

PERCY T. HERRING.

**THE RESPIRATORY MOVEMENTS OF THE THORAX AND (45) DIAPHRAGM.** Mosso, *Arch. ital. de Biol.*, T. xv., f. 1, 1903, p. 43.

THE subject matter of this paper, which records a series of very elaborate physiological investigations into the mechanism of respiration, falls into three sections: (1) The action of the nervous centres upon the movements of respiration. (2) The independence from one another of the rhythm and force of the movements, and of the tonus of the muscles of respiration. (3) The comparative physiology of the diaphragm and thorax.

1. His first experiments are directed to proving that carbonic acid gas, when it acts upon the respiration, must exert a stimulating action on the centres, not a reflex action through the sensory nerves of the lungs, because sensory impressions upon the skin and bones, however strong, will not produce the effect of carbonic acid upon an animal deeply under chloral.

The presence of cerebral centres he demonstrates by the marked and immediate action which psychical impressions in man show upon the respiratory tracings.

The effect of accumulated carbonic acid in the system, he found, varies widely in different individuals; some persons being able to hold the breath only for a few seconds, after which comes a violent reaction; others stopping the breath for one to two minutes, without much effect upon subsequent inspiration. These differences are independent of size, pulmonary capacity, etc., and are referred to a varied resisting power of the respiratory centres.

The generally accepted theory of Breuer and Hering, that changes in the volume of the lungs reflexly govern the movements of respiration through the sensory fibres of the vagus (dilatation of the lungs stopping inspiration and starting expiration, and their collapse *vice versa*), is controverted by his experiments. He found that on closing, by means of a mask, the nose and mouth in a large number of people, whether at the end of inspiration or of expiration, the resumption of breathing was almost always ushered in by an inspiration.

Again, tracings taken during respiration of hydrogen, show that deep inspirations of this gas produce apnoea similar to that on inhaling oxygen, and therefore the writer concludes that this apnoea is due not to the excess of oxygen but to diminution of carbonic acid.

2. The fact that the force of the respirations may increase and diminish while the rhythm remains constant, and *vice versa*, indicates that the mechanisms regulating the two are quite separate. The following eight combinations of change in rhythm and force are possible: (1)  $R >, F >$ ; (2)  $R <, F >$ ; (3)  $R <, F <$ ; (4)  $R >, F <$ ; (5)  $R$  constant,  $F >$ ; (6)  $R$  constant,  $F <$ ; (7)  $R <, F$  constant; (8)  $R >, F$  constant. Each of these is producible by various drugs and other agencies. In addition, the tonicity of the respiratory muscles may be depressed, or raised to tetanus independently of these, and the writer concludes from his experiments that the centre which maintains tonus lies in the spinal cord.

He insists that there must be a system of centres through the brain, medulla and cord, independent of one another, but controlled perhaps by the medullary centre. The respiratory movements he attributes to a periodic activity on the part of the nerve cells themselves comparable to the action of the heart; while the commonly accepted medullary centre he compares to the frog's brain, since in a pithed frog the most complex reflexes have their seat in the cord, and proceed without the government of the brain.

3. The comparative physiology of the diaphragm and thorax is treated by a record of elaborate experiments upon the functions of the two, but the results are still tentative. JOHN D. COMRIE.

## PSYCHOLOGY.

**THE EVOLUTION OF CONSCIOUSNESS.** W. H. B. STODDART, (46) *Brain*, Autumn 1903, p. 432.

The paper practically consists of two parts. The first part argues for the existence of sensation and consciousness of every cell of the organism. The second part shows especially by means of a diagram how sensations experienced by peripheral cells are transmitted to the complex colony of conscious neurons which constitute the cortex cerebri, this being the physical basis of mind.

It is first argued that such primitive organisms as the amoeba are sensitive and conscious. In higher animals special functions are assigned to special cells, *e.g.* muscle, but not to the total exclusion of other functions, *e.g.* sensation and excretion. All cells have some sensation, and hence, consciousness. The neuron is the most sensitive cell.

The sensations of peripheral cells (skin, rods and cones, cochlear hair cells) are first represented in bipolar cells (posterior root ganglia, internal granular layer of retina, ganglion spirale), and are re-represented in the cells of stations of divergence to the

cerebellum (nuclei gracilis and cuneatus, ganglionic layer of retina, auditory nucleus). The next representation is in basal ganglia (optic thalamus, external geniculate body, posterior corpus quadrigeminum), next in projection areas, and lastly in association areas.

In an addendum it is argued that there are centres of consciousness in the nervous system lower down than the cortex cerebri. For example, an ordinary skin-reflex is a conscious process on the part of one of these lower centres.

AUTHOR'S ABSTRACT.

## PATHOLOGY.

### DESCRIPTION OF AN ANENOEPHALIC (HEMIOEPHALIC), WITH

(47) CONTRIBUTIONS TO THE PHYSIOLOGY OF THE HUMAN CENTRAL NERVOUS SYSTEM. I. Anatomical. II. Clinical and Physiological. STEINBERG and LATZKO, *Deutsche Ztschr. f. Nervenheilk.*, Bd. 24, Heft 3 and 4, S. 209.

THIS paper consists of two parts, the first of which contains a resumé of the literature of anencephaly—or hemiccephaly, as the authors prefer to call it—and of the various conditions of the central nervous system, which have been described in different degrees of the condition. Following this, there is a detailed account of the anatomy of the central nervous system found in a case which came under their own notice, and which lived for about nine hours after birth.

The spinal cord, examined at several different regions, showed a condition almost identical with that described in a recent number of this review in the lumbar, thoracic, and cervical regions. The axis of the cord was continued up, and there was a fairly well-formed medulla and pons, from which the cranial nerves emerged as usual; but the fore-brain, mid-brain, and greater part hind-brain were absent; and the pyramidal tracts and Monakow's bundle were absent in the cord. The cerebellum was reduced to a vestige, and its connections were represented only by the restiform body, and this latter contained a small direct cerebellar tract from Clarke's column and uncrossed fibres from the nucleus cuneatus. The tracts found in the medulla were those of an ascending and endogenous nature.

The second part consists in a resumé of the clinical observations of a physiological and psychological character, which have been made on anencephalics which have for a longer or shorter time maintained an extra-uterine existence, of which the first dates from the year 1667, was made to the Royal Society, and "occasioned a question for the Cartesians, how motion could be

performed, and yet the glandula pinealis or corianum be wanting nor any nerves visible which come from the brain."

This interesting case lived for four days. Later communications have been directed to the solution of the same problem: how an organism whose central nervous system is so deficient can execute movements, as is undoubtedly the case.

The authors found that in their own case the child was capable of carrying on its circulation and respiration, and could move the muscles of the face, trunk and extremities; and they then discuss the position which the different centres for reflex movement must occupy, and the mechanism involved in their actions, as affected by the data found in their own observations.

DAVID WATERSTON.

#### **AN ALTERATION OF THE CORPUS CALLOSUM OBSERVED IN**

(48) **ALCOHOLIC SUBJECTS.** E. MARCHIAFAVA and A. BIGNAMI,

*Riv. di patol. nerv. e ment.*, Dec. 1903.

THE authors have observed a special morbid condition of the corpus callosum in three individuals who were known to have been alcoholics. The two cases that were observed clinically suffered, during the last few days of life, from a series of convulsive seizures, in the one case limited to the right side, in the other general. The lesions had essentially the same characters in each of the three cases. The whole of the extent of the corpus callosum was involved. On transverse section the tissues had a diffuse grey colour, excepting at the upper and under surfaces, which were normal in appearance. On microscopical examination they showed some rarefaction and increased vascularity; the vessel walls were not altered, excepting that some of the small arteries showed a zone of hyaline material, probably dependent upon degeneration of perivascular neuroglia fibres. The altered area contained chiefly a network of neuroglia fibres and axis-cylinders which had lost their myeline sheaths. In some preparations there were numerous granular cells. The degeneration of the medullated fibres was continued for a distance of a few millimetres into the white substance of the hemispheres and then ended abruptly. No morbid changes of note were discoverable in other parts of the brain. No evidence of secondary degeneration could be found.

The morbid process seemed to be one characterised mainly by degeneration of the myeline sheaths, with formation of granular cells and proliferation of neuroglia, the axis-cylinders being in greater part preserved. The authors incline to the view that the neuroglia change was the primary one. They think that it is certain that alcohol was the chief factor in the etiology of the lesions.

W. FORD ROBERTSON.

**BLOOD CHANGES IN DEMENTIA PARALYTICA.** A. R. DIFEN-  
(49) DORF, *Am. Journ. Med. Sc.*, cxvii., Dec. 1903, p. 1047.

D. FINDS that dementia paralytica is accompanied by a moderate and progressive anæmia, involving especially the hæmoglobin. The terminal stage of the disease is accompanied by a rise in the hæmoglobin, and erythrocytes, and a leucocytosis.

This leucocytosis begins earlier than the leucocytosis of the moribund state.

Paralytic attacks are accompanied by a leucocytosis—an observation which the author thinks is in favour of the theory of the toxic origin of the disease.

Leucocytosis is, however, absent in attacks of paretic excitement and stupor not terminal, as the toxic state is not intense enough to produce it.

Throughout the disease there is an increase of the polymorphonuclear leucocytes. This is most marked in the terminal stage.

Plasma cells were looked for but never seen in making the differential counts. Clinical records and blood counts from several cases are given and the literature is referred to.

ALEXANDER GOODALL.

## CLINICAL NEUROLOGY.

**TWO CASES OF ACUTE MYELITIS IN THE COURSE OF**  
(50) **SECONDARY CARCINOMA OF THE DURA MATER OR OF**  
**THE CORD.** G. BALLET and LAIGNEL-LAVASTINE, *Rev. Neurol.*,  
Dec. 15, 1903, p. 1120.

THE first case is one of cancer of the stomach, in the course of which the patient developed a flaccid paraplegia ten days before admission to hospital: the paraplegia had a gradual onset and was preceded by slight pains. On admission there was complete loss of patellar and Achilles reflexes, complete anæsthesia below a line a hand's breadth below the nipple, incontinence of fæces and retention of urine; œdema of the lower extremities, enlarged sub-clavicular and inguinal glands on both sides, distended superficial abdominal veins completed the clinical picture. Three weeks after the development of the paraplegia the patient died suddenly while being turned in bed.

The autopsy showed carcinoma of the stomach, which had propagated itself along the posterior chain of glands and invaded the vertebral bodies; an embolus in a dural artery had caused a secondary metastatic growth within the cord. On the posterior

aspect of the vertebral bodies from the third cervical to the fifth dorsal was a soft, whitish neoplasm without adhesion to the dura. The cord down to the fourth dorsal segment was normal. The fifth to the seventh dorsal segments showed acute myelitis with no invasion of carcinoma. The eighth dorsal segment showed nests of cancer cells, with patches of myelitis. Lower than the eighth segment no cancer cells were found.

The authors have found in the literature only three similar cases of spinal carcinoma secondary to visceral carcinoma.

The second case in this paper is one of generalised visceral carcinoma in which a similar flaccid paraplegia developed with total anaesthesia up to the umbilicus and paralysis of the sphincters.

The autopsy disclosed primitive carcinoma of the pancreas which had been propagated by the intervertebral foramina to the dura mater: there was external pachymeningitis of carcinomatous nature of the tenth and eleventh dorsal segments, acute diffuse myelitis extending from the twelfth dorsal to the third lumbar segment. The cord was not invaded by the cancer.

In both of these cases we have a visceral cancer; a subacute medullary or meningeal invasion without marked spinal symptoms; and then a sudden acute myelitis of toxic origin causing a flaccid paraplegia.

C. MACFIE CAMPBELL.

**HYSTERO-TRAUMATISM OR FOCAL HÆMORRHAGES WITH  
(51) PRINCIPAL SEAT IN THE BULB. LÉVI and MALLOIZEL,  
*Rev. Neur.*, Dec. 15, 1903.**

THE authors analyse carefully the symptoms presented by a patient after a fall on the left side of his head; while several symptoms suggested hysteria, there was much in favour of an organic lesion. The symptoms included anæsthesia of the right side, deafness of the left ear, spasmodic attacks of weeping, anæsthesia of the pharynx, globus hystericus, monocular diplopia on both sides, myoclonus; at the same time there was ankle clonus on both sides, and the patient's gait suggested an organic lesion.

The hemianæsthesia was of the right side and was not complete, sensibility to touch being almost intact, while that to heat, cold and pain was much impaired. Babinski and Nageotte have insisted on the presence of this dissociation in bulbar lesions, as well as of certain ocular troubles present in the case discussed—slight enophthalmos, myosis, narrowing of the palpebral aperture.

The authors also observed the presence of those symptoms upon which Babinski has laid stress in cerebellar lesions: a difficulty

in making a quick succession of movements (diadococinésie), difficulty in co-ordination, and a cataleptoid state of the muscles. While recognising a certain hysterical element in the case, the authors are of opinion that it is a case of an organic affection.

C. MACFIE CAMPBELL.

### **ENORMOUS TUMOUR OF THE POSTERO-PARIETAL REGION**

(52) **WEIGHING OVER HALF A POUND.** Absence of Localising Symptoms until late in the History of the Case; Operation; Death. F. X. DERCUM and W. W. KEEN, *Journ. of Nerv. and Ment. Dis.*, Dec. 1903, p. 737.

THIS patient presented the general symptoms of intracranial growth with double optic neuritis, also weakness of the right sixth nerve with slight muscular inco-ordination of the right side and astereognosis of the right sole. For a short period there was local pain and tenderness in the left post-parietal region. It was more than ten months after the onset of symptoms before definite localising signs appeared, namely, right hemiplegia, increased knee and ankle jerks, and extensor response associated with slight right-sided anæsthesia, astereognosis and hemianopia, with Wernicke's sign. Word-blindness was also present. The diagnosis having been made, the tumour, a sarcoma weighing 8 oz., was removed, the operation being completed in three stages. The patient died from shock.

In reviewing the history of the case the authors refer to the value of astereognosis and slight incoordination and the local tenderness as early localising signs. They conclude that the growth commenced subcortically and spread both upwards and downwards.

Dr Keen, who performed the operation, remarks on the great size of the tumour, on the vascularity of the scalp and bone over the growth, and also on the ineffectiveness of ligature of the common carotid to lessen the hæmorrhage from the brain. In this case one half of the superficial area of the false cerebri was exposed by the removal of the growth.

T. GRAINGER STEWART.

### **INFERIOR SYNDROME OF BENEDIOT.** COMBE, *Rév. mens. Mal. (53) de l'Enfance*, Jan. 1904.

THE patient was a little child four years old, who at the age of sixteen months had a severe attack of gastro-intestinal catarrh, which was followed by a complete ophthalmoplegia externa of the



left eye, with ptosis and external strabismus, developing very suddenly. Two months later, there followed paresis of the right arm and leg, becoming in a few weeks an advanced right-sided spastic paralysis. The eye condition seems at this time to have diminished somewhat. Finally, involuntary choreiform movements followed on the paralysed side. Two years later, the condition was as follows:—First and second cranial nerves intact both sides; third nerve, left, paralysis of external eye muscles, except the internal rectus, paralysis of the sphincter iridis, slight spasm of the orbicularis; right, paralysis of the inferior rectus and paresis of the inferior oblique; fourth nerve, paralysis of the superior oblique on the left side; sixth, spasmodic contracture of the left external rectus with hemichoreic movements; seventh, contracture of left facial muscle with hemichoreic movements also. The spastic paralysis of the right side of the body was more advanced, with incessant hemichorea; so that one might very fairly describe a crossed post-hemiplegic hemichorea, with hemiataxia, but without hemianæsthesia.

The exact diagnosis is gone into with the greatest care. It is sufficient to say that the author's conclusion is as follows:—Subacute poliomesocephalitis (of Wernicke), with two principal lesions, one in the peduncle, involving the nuclei of the third pair and of the fourth (left), the other in the pons at the level of emergence of the sixth and seventh (left) in the immediate neighbourhood of the left pyramidal tract. Add to this the trembling choreic movements of the paralysed parts, and it will be seen to be the syndrome of Millard Gubler (sixth and seventh), plus the syndrome of Benedict, plus an accessory peduncular lesion. The author proposes to call the combination of the two named syndromes the *inferior* syndrome of Benedict, as opposed to the ordinary or *superior* syndrome of Benedict (peduncular).

S. A. KINNIER WILSON.

**HYSTERIA IN CHILDHOOD.** BRUNS, *Jahrb. f. Kinderheilk.*, Bd. 58, (54) H. 6, Dec. 1, 1903, p. 895.

CHILDREN suffering from hysteria form about 2 per cent. of the patients seen in neurological practice, and about 20 per cent. of all cases of hysteria. Bruns includes cases occurring up to the age of 16, the average age being 10½ years, and by far the most cases occurring between the seventh and twelfth years. From the third to the sixth year he has only seen eight cases. He thinks it is improbable that the disease occurs, as French writers maintain, during the first two years of life. Boys and girls are almost

equally liable up to about the ninth year; thereafter most of the patients are girls, whose predisposition to hysteria steadily increases with the approach of puberty. Generally speaking, the worst cases occur in boys. Children who live in the country are quite as liable as those bred in the town; in fact, the most severe forms seem to occur principally among the former. Among the forms of hysteria most commonly met with in children, Bruns enumerates hemi- para- and monoplegia, with and without contractures and alterations of sensibility, joint neuralgia with contractures, aphasia, aphonia and mutism, blindness, spasms, varying from twitches of single muscles to chorea magna, tics, attacks of tachypnoea and dyspnoea, anorexia and vomiting, more rarely psychical disturbance, and not infrequently astasia abasia. The disease is usually monosymptomatic, that is, even if more than one hysterical manifestation be present, Charcot's stigmata of hysteria are almost always absent. Bruns explains this absence of stigmata (hystero-genetic zones, contraction of the visual fields, hemianæsthesia, etc.), by ascribing their presence in the adult, partially, at least, to suggestions arising from clinical examination. The adult, with a total paralysis of an arm, when the observer tests its sensibility, concludes that a paralysed limb ought not to feel, and in consequence anæsthesia develops. The comparative simplicity of the child's psychology does not, however, so readily admit of the same train of thought. This absence of stigmata renders the diagnosis difficult at times. The points on which the recognition of the disease is founded are gone into at some length by the writer, but as they are practically the same as in the adult they need not be repeated here. The hysterical symptoms in a child are prone to be both severe and complete—"massive," as the French say, and the patients react seriously to their morbid sensations. Thus paresis is much rarer than paralysis, aphasia than mutism, in which not only speech but phonation is in abeyance, and in hysterical neuralgia or headache the patient is more apt to throw himself wildly about than in similar non-hysterical affections. The prognosis is very favourable; of Bruns' 144 cases, 90 per cent. completely recovered; and as the only means of cure is found in suggestion, the sooner the diagnosis is made the better, since suggestion will be less readily responded to if other remedies have previously been unsuccessfully tried by the doctor. The first essential is to remove the child from its parents or friends to a suitable hospital or nursing home. Then comes the suggestion that, for example, a child with astasia abasia can walk, and Bruns lays great stress on the importance of taking the patient entirely by surprise in giving the order. The treatment should be carried out as soon as possible, and, if by any means this can be attained, completely at a single attempt. In other

cases, as, for example, hysterical fits, deliberate neglect of the symptoms is an important adjunct to suggestion.

J. S. FOWLER.

**HYSTERIA IN CHILDHOOD.** THIEMICH, *Jahrb. f. Kinderheilk.*, Bd. (55) 58, H. 6, 1903, p. 881.

THE author emphasises the monosymptomatic character of the affection at this age, the absence of stigmata, and the diagnostic importance of the rapidly curative action of suggestion. The monosymptomatic nature of hysteria is partly due to the simplicity of the psychology of the child, and partly due to the fact that in many cases the disease takes the form of one symptom arising in connection with a previous organic disease, *e.g.* hysterical constipation, requiring the use of enemata continuing for months after cure of the malady for which the enemata were originally required; hysterical diarrhoea with frequent small, normal motions as a sequel of gastro-intestinal catarrh; hysterical cough, hysterical vomiting, etc. In the same way, a fall or a short febrile illness may be followed by astasia abasia, or a slight injury to the arm by paralysis. Another common cause is imitation of their parents, as in a case of polydipsia and polyuria, which Thiemich saw in the child of a diabetic father. Besides imitation and auto-imitation, the author lays great stress on the nature of the child's training and the wisdom or unwisdom of the parents in dealing with trifling ailments and injuries as causes of hysteria. In addition, however, the children are nearly always neuropathic; they are frequently also delicate in other respects, and are not rarely neurasthenic—easily exhausted, with increased tendon reflexes, absence of the throat reflex, and high blood-pressure. That the environment is not all that is necessary to cause hysteria is shown by the fact that of two children of a family, both suffering from the same illness, one may become hysterical and the other escape. A predisposition to the disease must therefore exist, and that it is somewhat widely spread is shown by the occurrence of epidemics of hysteria in schools, in which half or more than half the children may be affected.

J. S. FOWLER.

**ON GASTRIC TETANY.** B. G. A. MOYNIHAN, *Boston Med. and Surg. Journ.*, Vol. cxlix., No. 19, Nov. 5, 1903, p. 591.

THE writer holds that the term "Gastric Tetany" should not be entirely restricted to the severer forms of the disease, but should

include all cases of tetany and tetanoid spasms associated with and directly due to diseases of the stomach.

He also believes that the disease in a more or less mild form is a much commoner condition than has previously been supposed. He gives a general review of the symptoms and course of the disease, and states the more recent views as to its causation, pathology and treatment. The theory put forward by Küssmaul to the effect that the disease is due to general desiccation of the tissues of the body has now been practically abandoned, and the author thinks it possible that the true solution of the difficulty may be found in a combination of the reflex and auto-intoxication theories.

With regard to the pathology of gastric tetany he notes the now generally accepted fact that dilatation of the stomach is practically invariably present, this being usually due to stenosis at or near the pylorus, resulting from partial or complete healing of a chronic ulcer of the stomach or duodenum. In the treatment of the condition he emphasises the necessity of frequent and thorough lavage, combined with rigid dieting and the administration of sedatives when necessary, and he also refers in detail to the method of treatment adopted by Professor Greenfield in a case recorded by Carnegie Dickson in *The Practitioner*, vol. i., 1903. The final and the appropriate treatment in all cases of gastric tetany should, however, be surgical, and recourse should be had as soon as possible to suitable operative interference, such as gastro-enterectomy, etc., according to the nature of the pathological lesion present, several very successful cases so treated having been recently recorded.

W. E. CARNEGIE DICKSON.

#### THE MOVEMENTS OF SUPERIOR INTERCOSTAL MUSCLES IN

(57) **HEMIPLEGICS.** PIERCE CLERK, *Am. Journ. Med. Sc.*, Dec. 1903, p. 1029.

THE writer quotes the opinions of Gowers and of Hughlings Jackson upon this subject. The former made the general statement that in hemiplegics the two sides of the thorax move equally in ordinary respiration; but if the patient takes a deep breath and brings into action the extraordinary muscles of respiration, the half of the thorax on the paralysed side often expands less than the other. Jackson in 1895 pointed out that in ordinary respiration the action of the superior intercostals was greater during quiet inspiration on the paralysed than on the sound side. He recorded twenty-eight cases of hemiplegia, in eighteen of which he had met with this experience.

The writer confirms the latter opinion as the result of examining

161 cases of hemiplegia, without regard to age, character, duration or severity, and finding in all the double change of increased movement during quiet and less movement during forced inspiration on the paralysed side, as compared with the sound side.

He offers, as explanation of this phenomenon, the theory that in lesions of the internal capsule causing paralysis, the cortical inhibition over the medullary respiratory centre on the paralysed side is destroyed more than on the other.

He considers the relation of these facts to Spencer's work on the existence of an inhibition centre for respiration situated in the cortex just outside the hinder end of the olfactory tract.

Finally he states that the principle underlying the respiratory sign is wide in its application, being discoverable also in the action of the 5th (third division), 7th, and 9th cranial nerves.

JOHN D. COMRIE.

#### **NOTE ON THE RESPIRATORY MOVEMENTS IN HEMIPLEGIA.**

(58) JUDSON S. BURY, *Lancet*, Dec. 19, 1903, p. 1714.

THE writer refers to the statement of Hughlings Jackson mentioned above, and confirms it for 60 per cent. of his own cases. He also includes in his article a diagram of tracings from the two sides of the thorax, which show graphically the extent of the difference in respiratory movements.

JOHN D. COMRIE.

#### **A CONTRIBUTION TO OUR KNOWLEDGE OF THE HEMI-**

(59) ANOPIA IMMOBILITY OF THE PUPIL. FRIEDLÄNDER and KEMPNER, *Neurolog. Centralbl.*, Jan. 1904, p. 2.

THE difficulty in eliciting Wernicke's sign is well known, and Dr Kempner along with Von Fragenstein in 1899 devised and described a special instrument with which small portions of the retina can be illuminated while the diffusion of light over the remainder of the retina is reduced to a minimum. The instrument takes the form of a short cylinder containing a small electric light, a convex lens and a very small aperture of exit for the rays. With this instrument the authors have been able to demonstrate the presence of Wernicke's sign in the clearest possible fashion in a case of typical left side homonymous hemianopsia. From the history and the accompanying symptoms in this case, viz., left partial hemiplegia, hemiatrophy of the tongue, paresis of the palate, difficulty in swallowing, headache, mental impairment, etc., a diagnosis

was made of brain syphilis with an involvement of the right optic tract. The presence of considerable optic atrophy is in conformity with this diagnosis since in hemianopsias from centrally placed lesions no change takes place in the appearance of the discs.

It does not appear to be clearly stated in this article whether Wernicke's sign could be elicited in the case quoted by the methods usually employed, or whether the employment of this instrument, "Pupillenreactionsprüfer," demonstrated the presence of the symptom when other means had failed.

J. V. PATERSON.

#### **A MOVEMENT OF THE EYEBALL ACCOMPANYING THE**

**(60) PUPILLARY LIGHT REACTION.** FREUND, *Prag. med.*

*Wchenschr.*, Bd. xxviii., Nr. 44, 1903, p. 569.

THE case recorded by Dr Freund as showing this anomalous associated movement was that of a girl æt. 20 years, who in March 1900 had sustained a severe head injury from a fall. After the accident the left eye was found to be blind and gradually deviated outwards. The cause of blindness was optic nerve atrophy due to involvement of the nerve in a fracture of the base of the skull. The direct light reaction was naturally absent in this eye and a prompt consensual reaction was present as is usual in these cases. On shading the right eye the left pupil dilated consensually and the left eyeball turned downwards to an amount equal to about  $\frac{1}{3}$  of the height of the cornea. On re-exposing the right eye to the light, the left eyeball moved upwards to its former position. The movements of the left eyeball occurred somewhat slowly, the pupillary movements preceding them by an appreciable interval. The right eye remained quite stationary all the time. Instillation of a mydriatic into one or both eyes did not affect the movements of the left eye in the least degree. The pupillary movements of the right eye related to accommodation and convergence did not call forth the movement, which was thus shown to be intimately associated with the light reaction. The amount of movement it may be noted, was greatest with the right eye looking straight forwards, less when the eyes were turned to the left, and hardly observable when the eyes were turned as far as possible to the right. In seeking for an explanation of the condition, Dr Freund agrees with the opinions of Lewinsohn and Amat, who described a similar case. He supposes that the stimulus proceeded from the reflex centre, and that this centre having been injured, the stimulus more readily spread along abnormal paths.

J. V. PATERSON.

**ON SECONDARY CONTRACTURE OF THE LEVATOR PALPE-  
(61) BRÆ SUPERIORIS IN THE COURSE OF FACIAL PARA-  
LYSIS.** E. BERGER and R. LOEWY, *Rev. Neur.*, Dec. 15, 1903.

THE authors give the cases of three patients with this affection : in the first case the facial paralysis was due to a peripheral neuritis, in the second to a basal hæmorrhage, in the third case it came on after an operation for cancer in the parotid region.

The symptoms are the following : the palpebral aperture is widened, the border of the upper eyelid is higher than on the unaffected side ; there is presence of Stellwag's and of Von Graefe's sign ; the patient cannot shut the eye on the affected side, but if one depress the upper eyelid and maintain it in position for a minute or two the patient can then close his eye, at least partially.

The authors are of opinion that the signs of Stellwag and of Von Graefe in exophthalmic goitre are due to the secondary contracture of the levator palpebræ superioris consecutive on the continued paresis of the orbicular.

C. MACFIE CAMPBELL.

**THE CORNEOMANDIBULAR REFLEX.** VON SÖLDER, *Neurolog.*  
(62) *Centralbl.*, 1902, p. 111.

**ON THE CORNEOMANDIBULAR REFLEX.** J. KAPLAN, *Neurolog.*  
(63) *Centralbl.*, 1903, p. 910.

**THE CORNEOMANDIBULAR REFLEX.** VON SÖLDER, *Neurolog.*  
(64) *Centralbl.*, 1904, p. 13.

THE first paper contains the description of a reflex movement of the jaw to the opposite side on touching one cornea. It is, of course, associated with the usual corneal reflex.

It consists in a purely lateral movement of the mandible, due to contraction of the external pterygoid of the side on which the cornea is touched, and to obtain it, it is necessary that the teeth should be slightly separated.

It is by no means always present even in health, and no facts are given as to its occurrence or absence in disease, but it is noteworthy that it occasionally persists in coma when the corneal reflexes are absent.

The author believes that it should be classed among the super-

ficial reflexes, and that its path is along the sensory and motor divisions of the trigeminus.

In the second paper, Kaplan suggests that the contraction of the external pterygoid is synergic with the action of the orbicularis palpebrarum, and is not a true reflex action.

V. Sölder answers that Kaplan's objections are baseless, and further supports his theory by the facts that: (1) contraction of the pterygoid only accompanies contraction of the orbicularis obtained by touching the cornea; (2) the phenomenon is unilateral, though the associated corneal reflex is always bilateral; and (3) the two reflexes may be disassociated in coma. GORDON HOLMES.

**A NEW SPINO-MUSCULAR PHENOMENON IN NORMAL PER-  
(65) SONS.** D. MC CARTHY, *Neurolog. Centralbl.*, 1904, p. 16.

STRONG contraction of the semi-tendinosus and semi-membranosus results from percussion on the second and third lumbar spines. It is best obtained by making the patient lie in the prone position with the lower extremities limp.

It is probably of the same nature as Bechterew's "lumbo-femoral reflex," and is probably not a true reflex.

GORDON HOLMES.

## Reviews

**ALLGEMEINE ANATOMIE UND PHYSIOLOGIE DES NERVEN-  
SYSTEMS.** A. BETHKE. 480 pages. Two plates, 95 illustrations. George Thieme, Leipzig, 1903. M.13.50.

It is long since neurology received such a valuable contribution as the noteworthy book under review. Its aim is not to be regarded as a text-book on the subject of which it treats, but rather to be a treatise on the structure and physiology of the nervous system as indicated by the most recent researches.

It is scarcely necessary to say that the work of this author accumulates all the available material that argues against the validity of the so-called neurone-theory, but even those who have the least sympathy with his chief aim cannot but admire the logical and forcible statement of his case, and even more the bulk of original work his volume contains. Indeed, to quote the words of the preface, "original thoughts and facts are now so constantly made public in journals and transactions of learned societies, that



it is with more or less surprise one finds such in book form, apart from monographs," yet fully half of these 500 pages are devoted to the description of the author's own work, much of which is new, while that already published is further expanded and elaborated.

As might be expected from the co-worker of Apáthy and Nissl in the finer structure of nerve-cell and fibre and the demonstration of continuity, as in contrast to contiguity, in the nervous system, fully a quarter of the pages deal with the minute histology of cell and fibre, and these contain the matter which will be perhaps of most general interest.

As is known, Apáthy and Bethe have already some years ago demonstrated the existence in invertebrates of continuous and unending neurofibrils which remain unbranched and individual in the axis-cylinders of nerves, but split up, branch and anastomose with one another in the special end organs, in the ganglion cells with which they enter into relation, and in the neuropile, the more or less structureless mass in which the ganglion cells are embedded.

As neurofibrils can be found everywhere where there is evidence of nervous function, they are regarded as the essentially specific constituent of the nerve; and as they connect cell with cell and anastomose without the cells, there is complete relative structural continuity of all parts of the nervous system of the invertebrates.

Apáthy's work did not include the vertebrate animals, and the little we know of the "essentially specific constituent" of their nervous system is chiefly due to Bethe's earlier publications. A short resumé of his latest conclusions cannot fail to be of interest.

Here, as in the invertebrates, the axis-cylinders contain individual unbranching fibrils embedded in a peri-fibrillar substance, and Bethe seems to have conclusively demonstrated that they are the only constituents of the axis-cylinders which are not interrupted at Ranvier's nodes, and they must, he argues, consequently be regarded as the conducting element. Their arrangement in the cells is, however, quite different from that in the invertebrates, for the nerve-fibrils pass from dendrite to dendrite and from dendrite to axite unbranched, and maintaining their individuality. The only exceptions which have been met are the cells of the spinal ganglia and of the lobus electricus of the torpedo, in which the nerve-fibrils anastomose among themselves to form a net (*Fibrillengitter*).

The connection of cell with cell is brought about by the arborisation of axis cylinders round the cell and its dendrites (*Achsenzylinderhose*). This arborisation is not terminal, as some of the branches anastomose among themselves, and others are indirectly continuous through a pericellular structure (*Golginetz*), first de-

scribed by Golgi as a neurokeratin envelope, and was mistaken by Apáthy for a glial sheath. It is a fine-meshed net which envelops the whole of the cell body and its dendrites, and though probably of different constitution from the nerve-fibrils, these latter, where they come to the surface in either the cell or its dendrites, run into and fuse with it. Thus results the continuity of the intracellular fibrils with those of the *Achsenzylinderhose*. The Golgi-net is thus the intermediary between the cell and the axis-cylinders which split up round it. Further, this intermediary substance is probably that postulated by Nissl, as his "Grau," which in the cortex, at least, lies between the cells, and is not as in other regions limited to them as their enveloping sheath, but extends through the grey matter in which they lie.

Of considerable interest, too, is the study of the so-called peripheral nerve-net through which in molluscs the innervation of the muscles takes place, and which is the mechanism of the majority of their simpler reflexes. This net is directly continuous through their body, and through it a reflex movement of the whole musculature can be brought about by local stimulation of sufficient intensity. A similar nerve-net is present in the cardiac musculature of the frog. It can only be completely interrupted by complete section of the muscle, and it is through its fibres, and not from muscle fibre to fibre, that the impulses pass.

Several pages are devoted to the discussion of nerve degeneration. The author adds new observations on the neurofibrils after nerve injury, showing that granular degeneration of them is the earliest morphological change, and synchronous with which is loss of physiological inductibility. Further, morphological degeneration is due to local trauma of the nerve and not to separation from a trophic centre. The discussion on nerve-regeneration is lengthy and full. Complete autogenous regeneration of the peripheral part which was prevented from uniting with the central stump, is confirmed by numerous original experiments. The regenerated part may, in young animals at least, even become capable of conduction. Further experiments have confirmed Bethe's previous observation, that when an autogenous regenerated nerve is again sectioned, only its peripheral part degenerates, though the central have no connection with any such trophic centre as a ganglion cell. Further, regenerated nerves unconnected with their cells will after a time become incapable of conduction, and their fibrils can be no longer primarily stained.

The regeneration of nerve commences by proliferation of the nuclei of Schwann's sheath and increase and fusion of the protoplasm to form a continuous band; in the latter nerve-fibrils develop in the neighbourhood of the nuclei, and extending longitudinally fuse at the site of the future Ranvier's nodes. Regeneration of nerve

fibres in the dorsal columns of the spinal cord was found after section of the dorsal roots. Other conclusions on this subject were: that the peripheral end of one nerve can unite with the central of another; that a piece of one nerve can be grafted into another, provided that the "polarisation" of the former is respected, i.e. that its central end is centrally directed; that the central ends or peripheral ends of nerves cannot grow together; and that at least a trophic (? functional) connection can take place between a motor and a sensory nerve.

By original investigations it is attempted to prove that peripheral nerves do not develop as a process of a central cell, but from a chain of spindle cells (? origin) round each of whose nuclei nerve-fibrils are differentiated, and this is regarded as a strong argument against the existence of the neurone as a nervous unit.

The latter half of the book discusses the physiology of the nervous system, and in it the author's original work takes no less a share than in the anatomical part. The effort to explain nervous processes by, and correlate them with, the doctrine of continuity, is everywhere apparent, and whether the attempt is received with approval or not, the methods are admirable. The nerve-cells lose their hitherto recognised importance in contrast to the conducting fibres and their constituent neuro-fibrils, and the function of conductivity in the latter depends on the presence of a substance named fibrillic acid (*Fibrillensaure*), which, in contrast to the tigroid, is soluble in acidulated alcohol.

On stimulation of a nerve by the galvanic current this fibrillic acid passes from the anode to the kathode, and this is the explanation the well-known reduction of irritability in the neighbourhood of the anode. Narcotics act by inhibiting the power of movement of this substance.

In other chapters, reflexes, summation, tone, inhibition and rhythmical movements are fully discussed.

Finally the book can be thoroughly recommended to all not quite fettered by conventional opinions and theories, and to those who welcome fresh ideas and new views.

GORDON HOLMES.

**LES NERFS DU CŒUR CHEZ LES TABÉTIQUES.** Par le Dr  
JEAN HEITZ, *Thèse de Paris*, 1903, pp. 217.

THE nucleus round which this monograph has grown, consists of a histological examination of the cardiac innervation of twelve cases of *tabes dorsalis*, together with an account of the clinical history of these. The better to elucidate the pathological conditions found,

the author devotes the first portion of the book to a survey of the anatomy of the cardiac plexus both sympathetic and pneumogastric, and makes mention of the original work of many who have contributed to our knowledge of this still obscure subject. It is perhaps unnecessary that mention should be made of too many foreign authorities in such a preliminary sketch, but we should have thought it scarcely possible that justice could have been done even to an imperfect anatomical bibliography, without some reference to the well-known work of Gaskell in this sphere. The author would appear to be unaware of its existence. With the exception of this omission the account given is sufficiently full and instructive for a preface to the succeeding sections.

Osmic acid and carmine were the chief stains used in the examination of transverse sections of the nerves, while the cells of the ganglia were also stained by Nissl's method. The object of the examination of the nerve sections was to determine the number of large and small myelin-sheathed fibres in the peripheral cardiac innervation. The enumeration of these was made under the microscope by means of a cover-glass engraved in squares as for blood-counts. Having obtained approximately correct standards, sections similarly stained from the pneumogastric and sympathetic innervation of tabetics was compared with these. The net result was to discover that in tabetics the myelin-sheathed fibres, both large and small, were less numerous than under normal circumstances. There was also in some cases a general sclerosis of the nerve trunks, but this was not the rule. Dr Heitz appears to have found the sympathetic changes to have been more constant than the pneumogastric. While changes in the cardiac plexus and cervical sympathetic appear usually to have been related, the connection of these with those in the spinal roots was less evident. The sympathetic ganglia, according to the author, were little altered, and changes in them chiefly pigmentary, and less certainly attributable to the specific poison. Two good coloured plates are given, showing the rarefaction of the myelin fibres already mentioned, and which the author regards as pathological.

Having sketched the anatomy and physiology of cardiac innervation and related his own pathological investigations, he next applies these in endeavouring to explain some anomalies of cardiac action and vascular states met with in tabetics. In considering the pathogenesis of aortitis in these cases he, however, rejects the trophic hypothesis of Teissier and his followers, and maintains with most observers that both arterial and nervous lesions are directly due to the specific poison. The latency or absence of symptoms in syphilitic aortitis, Dr Heitz considers to be due to a form of cardiac analgesia resulting from the neural changes in the sympathetic mentioned. Space prevents our discussing more at length the

author's conclusions as to the pathogenesis of certain other disturbances, such as tachycardia and cardiac pain. On these points he is, we consider, wisely inconclusive in his remarks, but his final position is reached after careful argument. The monograph closes with a detailed account of the clinical history and post-mortem examination of the twelve cases on which the work is mainly based. The investigation was originally suggested by Merklen and carried out in his laboratory and that of Professor Dejerine, and is to be recommended as a sound contribution towards the better knowledge of a much neglected field of enquiry in visceral disease. For the acceptance of the author's conclusions, further work on the same lines is required, to exclude possible fallacies due to the level at which the nerve sections have been made and to the comparatively small number of cases examined; and also, to control the conditions found in tabetics, by those occurring in a larger number of persons not so affected. But we sincerely congratulate the author on having done careful work in a much needed direction.

ALEXANDER MORISON.

---

### Obituary Notice.

#### GEH. MED.-R. PROF. DR FRIEDRICH JOLLY OF BERLIN.<sup>1</sup>

On the 4th of January of this year Professor Friedrich Jolly of Berlin died suddenly and unexpectedly. From the midst of his active scientific and practical career as professor in the University, as director of the psychiatric and nervous clinique of the Königlichen Charité, and as dean of the medical faculty in Berlin, he was called away by a death which, on account of his hitherto perfect condition of health, seemed at first almost incomprehensible, and which has deeply affected all who were connected with him, his nearest relatives, his colleagues, his students, and the whole scientific world. Psychiatry and neurology especially have lost in him one of their most prominent representatives.

Friedrich Jolly was born in Heidelberg on the 24th November 1844, his father being the well-known physicist, Philipp Jolly. When the latter was called to Munich in 1855, young Jolly entered the Gymnasium there, and in Munich he began his medical studies in 1862. Justus von Liebig, Siebold, Nägeli, Pettenkofer, Nussbaum, Bischoff, and others were his teachers. After studying for two terms in Göttingen, and producing in the Physiological Institute there under Meissner a work on "Succinic

<sup>1</sup> The Editor's thanks for this obituary notice are due to Dr W. Seiffer, Privat-dozent an der Universität und Oberarzt an der Nervenkl. der Kgl. Charité, Berlin.

Acid in the Urine," he completed his studies in Munich, and graduated there with a thesis "On the Ganglion-cells of the Spinal Cord." Thus, even as a student he showed a strong scientific interest in the central nervous system. From 1866 to 1868 he was clinical assistant in the General Hospital of Munich under Pfeuffer. After passing the State examination with distinction in 1868, he went to study in Vienna and Berlin. In Berlin, where he happened to arrive on the day of the death of Griesinger, whose successor he was later to become, he made the acquaintance of Westphal, Griesinger's successor, as well as of v. Gräfe, Virchow, and others, under whom he further pursued his studies. Meynert's psychological investigations had attracted him to Vienna, and there, along with Meynert and the well-known pathologist Stricker, he worked upon traumatic inflammation of the brain; whilst he also studied electrotherapy with Benedict.

In 1869 he was assistant in the Wernick Lunatic Asylum in Bavaria, along with Gudden, the future Munich alienist. In the following year he went to be assistant to Rinecker in the Psychiatric Clinique in Würzburg. At Würzburg his intimate friendship with von Recklinghausen and A. Pick commenced. He qualified at this University as privatdocent with a work "On the Intracranial Pressure and the Circulation of the Blood in the Skull." From Bavaria he proceeded on a visit to France and England, with a view to learning the scientific methods of dealing with the insane in use in these countries. He was particularly interested in the so-called "family care of the insane" in Scotland, upon which he published a report after his return to Germany.

Two years later Jolly was called to succeed Krafft-Ebing at the University of Strassburg. Here he not only won a great reputation and universal love and honour both as a physician and a teacher, but he also drew up the plans for a beautiful new building for the Strassburg Psychiatric Clinique, which he was able to open in 1886. In Strassburg he applied himself not only to the teaching of psychiatry, but gave very special attention to the teaching of nervous diseases; indeed, he organised a polyclinique for that special purpose.

During the year 1870 he had received calls from the Universities of Leipzig, Heidelberg and Würzburg, all of which he had declined. It was only in 1890, when on Westphal's death the University of Berlin invited him to fill this important chair, that he accepted the call, and here for thirteen years he has carried on an uninterrupted work as director of the large combined clinique for nervous and mental diseases, together with the polyclinique for nervous diseases, as one of the most beloved teachers of academic youth, as President of the "Berliner Gesellschaft für Psychiatrie und Nervenkrankheiten," as one of the directors of

the "Vereins für Psychiatrie in Berlin," of the "Deutschen Vereins für Psychiatrie," of the "Gesellschaft der Charité-Ärzte," and other scientific societies. He was also a member of the "Wissenschaftlichen Deputation für das Medicinalwesen in Preussen," a member of the "Reichs Gesundheits-Amtes," and editor of the *Archives für Psychiatrie und Nervenkrankheiten*.

When the reconstruction of the "Königlichen Charité-Krankenhaus" was commenced, one of the first buildings to be constructed was the new psychiatric and nervous clinique, which was erected from Jolly's plans, and is now almost completed.

The extraordinary number of publications to which his scientific work gave rise are scattered among various medical journals; the best known are his classic work "Über die Hysterie und Hypochondrie" (1877), his investigation "Über den elektrischen Leitungswiderstand des menschlichen Körpers" (1889), his lecture upon "Irrthum und Irrsinn" (1894), his contribution to the pathology and nosology of what he has called "Myasthenia pseudo-paralytica," "Zur Korsakoff'schen Psychose," his monograph "Über die funktionellen Neurosen" (1900), etc.

The construction of the psychiatric and nervous clinic in Berlin, to which in his later years he devoted a great amount of time and labour, and which for years to come will remain a model institution, he was not to live to see fully completed. Early in 1904, the year in which this building was to be opened for use, and in which Jolly was to have been presented by his students with a "wissenschaftliche Jubiläumsgabe" to celebrate his 60th birthday, he died from an affection which he had had indeed for a long time, but which had caused him no trouble, namely, an aneurism of the ascending aorta, which by perforation into the pericardium brought about his lamented death.

## Bibliography

### ANATOMY

- ANASTAS SHUNDA. Localisation cérébrale du nerf pneumogastrique. Bukarest, 1903.
- KURT GOLDSTEIN. Die Zusammensetzung der Rückenmarkshinterstränge. *Monatschr. f. Psychiat. u. Neurol.*, Bd. 14, H. 8, S. 401.
- G. ELLIOT SMITH. Note on the Communication between the Musculo-spiral and Ulnar Nerves. *Journ. Anat. and Physiol.*, Jan. 1904, p. 162.
- G. ELLIOT SMITH. The Fossa Parieto-Occipitalis. *Journ. Anat. and Physiol.*, Jan. 1904, p. 164.
- G. ELLIOT SMITH. Note on an Exceptional Human Brain presenting a Pithecoïd Abnormality of the Sylvian Region. *Journ. Anat. and Physiol.*, Jan. 1904, p. 158.
- MOTTA COCO e LOMBARDO. Contributo allo studio della struttura e delle granulazioni fucsinofile. *Riv. mens. de. psichiat. forense*, Nov. 1903, p. 443.

## COMPARATIVE ANATOMY—

- MAX WOLFF. Das Nervensystem der polypoiden Hydrosco und Scyphosco. *Zeichr. f. allg. Physiol.*, Bd. 8, H. 3, S. 191.
- DAVID HEPBURN and DAVID WATERSTON. A Comparative Study of the Grey and White Matter of the Motor Cell Groups and of the Spinal Accessory Nerve in the Spinal Cord of the Porpoise. *Journ. Anat. and Physiol.*, Jan. 1904, p. 105.
- MISCH. Das Binnennetz der spinalen Ganglienzellen bei verschiedenen Wirbelthieren. *Inaug.-Dissert.*, Berlin, 1908.
- OWSJANNIKOW. Das Rückenmark und das verlängerte Mark des Neunanges. Voss, Leipzig, 1908, M. 2.

## PHYSIOLOGY

- R. AMABILINO. Sulle prime vie olfattive. *Riv. Speriment. di Freniatria*, vol. xix., f. iv., 1908, p. 816.
- SHINKICHI IMAMURA. Ueber die corticalen Störungen des Sehactes und die Bedeutung des Balkens. *Arch. f. d. ges. Physiol.*, Bd. 100, H. 9-10, S. 495.
- DONAGGIO. Per il problema dei rapporti tra vie di conduzione intercellulari e periferia della cellula nervosa. *Riv. Speriment. di Freniatria*, vol. xix., f. iv., 1908, p. 825.
- MOSSO. Azione dei centri spinali sulla tonicità dei muscoli respiratori. *Gior. d. r. Accad. di med. di Torino*, vol. lxvi., N. 11, 1908, p. 755.
- MUNK. Über die Folgen des Sensibilitätsverlustes der Extremität für deren Motilität. Reimer, Berlin, 1908, M. 2.
- The Physiology of Fatigue. Leading Article, *Brit. Med. Journ.*, Jan. 16, 1904, p. 145.
- GIULIO OBICI. Influenza del lavoro intellettuale prolungato e della fatica mentale sulla respirazione. *Riv. Speriment. di Freniatria*, vol. xix., f. iv., 1908, p. 689.
- LINDSTÄDT. Neuere Forschungen über die Verrichtung der Schilddrüse, ihre Beziehungen zum Kropf, Kretinismus, Epilepsie, etc., Studien auf dem Gebiete der Nervenphysiologie und Pathologie sowie des Blutlebens. Fischer, Berlin, 1904, M. 1.50.
- MERZBACHER. Untersuchungen an winter-schlafenden Fledermäusen. II. Mittheilungen. Die Nervendegeneration während des Winter-schlafes. Die Beziehungen zwischen Temperatur und Winterschlaf. *Arch. f. d. ges. Physiol.*, Bd. 100, H. 11-12, S. 586.
- M. et MME. LAPICQUE. Recherches sur la loi d'excitation électrique. *Journ. de Physiol.*, T. v., No. 6, p. 991.
- BRÜNNIGS. Beiträge zur Elektrophysiologie. II. Mittheilung. Ueber Ruhestrom und Reizung. *Arch. f. d. ges. Physiol.*, Bd. 100, H. 7-8, S. 367.
- WEDENSKY. Le téléphone comme indicateur de l'excitation du nerf, à propos des objections faites par M. Tahirco. *Journ. de Physiol.*, T. v., No. 6, p. 1042.
- FLORA. Il tetano faradico nello studio di alcuni fenomeni nerveo-muscolari. *Riv. crit. di clin. med.*, Anno iv., No. 51, 1903, p. 801.
- MANIZER. Stoffwechselstudien über den Einfluss geistiger Tätigkeit und pathologische Anatomie dieser Erkrankung. *Monatschr. f. Psychiat. u. Neurol.*, Bd. 16, H. 6, S. 460.
- J. BEARD. A Morphological Continuity of Germ-Cells as the Basis of Heredity and Variation. *Rev. Neurol. and Psychiat.*, Jan. 1904, p. 1.
- CARLO CENI. Effetti della tireodectomia sul potere di procreazione e sui discendenti. *Riv. Speriment. di Freniatria*, vol. xix., f. iv., 1908, p. 837.

## PSYCHOLOGY

- PIÉRON. Le temps d'association simple. *Rev. de Psychiat.*, Dec. 1903, p. 545.
- MESSMER. Zur Psychologie des Lesens bei Kindern und Erwachsenen. *Arch. f. d. ges. Psychol.*, Bd. 2, H. 2-3, S. 190.
- BRADLEY. A Definition of Will. *Mind*, Jan. 1904, p. 1.
- HOHENEMSER. Versuch einer Analyse der Scham. *Arch. f. d. ges. Psychol.*, Bd. 2, H. 2-3, S. 299.



- FAIRBROTHER. The Relations of Ethics and Metaphysics. *Mind*, Jan. 1904, p. 88.  
 WALSH. Kant's Transcendental Idealism and Empirical Realism (ii.). *Mind*, Jan. 1904, p. 54.  
 OGDEN. Untersuchungen über den Einfluss der Geschwindigkeit des lauten Lesens auf das Erlernen und Behalten von sinnlosen und sinnvollen Stoffen. *Arch. f. d. ges. Psychol.*, Bd. 2, H. 2-3, S. 93.  
 STOLL. Suggestion und Hypnotismus in der Völkerpsychologie. Veit & Co., Leipzig, 1903.

## PATHOLOGY

- R. T. WILLIAMSON. Changes in the Spinal Cord in Diabetes Mellitus. *Brit. Med. Journ.*, Jan. 16, 1904, p. 122.  
 GEORGE LAMB and WALTER K. HUNTER. On the Action of Venoms of Different Species of Poisonous Snakes on the Nervous System. *Lancet*, Jan. 2, 1904, p. 20.  
 BABONNELX. Les paralysies ascendantes aiguës dans la diphtérie expérimentale. *Arch. gén. de méd.*, Dec. 22, 1903, p. 3201.  
 LESTELLE. Des accidents cérébraux consécutifs à la ligature de l'artère carotide primitive. Roussel, Paris, 1903.

## CLINICAL NEUROLOGY AND PSYCHIATRY

## GENERAL—

- SUCKLING. On Movable, or Dropped Kidney: its Relation to Diseases of the Nervous System. H. K. Lewis, London, 1903.

## MUSCLES—

- SCHIEFFERDECKER. Beiträge zur Kenntnis der Myotonia congenita, der Tetanie mit myotonischen Symptomen, der Paralysis agitans und einiger anderer Muskelkrankheiten, zur Kenntnis der Aktivitäts-Hypertrophie und des normalen Muskelbaues. Mit klinischen Beiträgen von Schultze. *Deutsche Zechr. f. Nervenh.*, Bd. 25, H. 1-4, 1904, S. 1.

## PERIPHERAL NERVES—

- Sciatica. Leading Article. *Brit. Med. Journ.*, Jan. 16, 1904, p. 146.  
 HOWARD DISTIN. A Note on "Brachial Neuralgia." *Brit. Med. Journ.*, Jan. 16, 1904, p. 124.

## SPINAL CORD—

- Tabes.—ERB. Syphilis und Tabes. *Berl. klin. Wchnschr.*, Nos. 1-2, 1904.  
 HARTMANN. Ueber Tabes juvenilis und Lues hereditaria. *München. med. Wchnschr.*, Dec. 22, 1904, S. 2264.  
 SIMONELLI. Sulla questione delle affezioni così dette "parasifilitiche." *Clin. Med.*, Nov. 4 e 18, 1903, p. 620.  
 PAL. Die vasomotorischen Begleiterscheinungen der langsamierenden Schmerzen und das Alternieren tabischer Krisen. *Wien. med. Wchnschr.*, Jan. 2, 1904, S. 2.  
 GRAETZER. Tabische Osteoarthropathie der Wirbelsäule. *Deutsche med. Wchnschr.*, Dec. 24, 1903, S. 992.  
 FRENKEL. The Treatment of Tabetic Ataxia. Rebman, Ltd., London, 1903.  
 KOUNDJY. Die Massage bei Tabes dorsalis. *Ztschr. f. diät. u. physikal. Therapie*, Bd. 16, H. 10, 1904, S. 536.  
 Disseminated Sclerosis.—VORBERG. Zur klinischen Differentialdiagnose der Sclerosis multiplex cerebrosplanialis. Speyer & Kaerner, Freiburg, 1903, M. 0.80.  
 STRÄHUBER. Bemerkungen zu der Arbeit des Hrn. Bielschowsky: Zur Histologie der multiplen Sklerose. *Neurol. Centralbl.*, Jan. 16, 1904, S. 66.

MAX BIELSCHOWSKY. Die marklosen Nervenfasern in den Herden der multiplen Sklerose. *Neurol. Centralbl.*, Jan. 16, 1904, S. 59.

#### BRAIN AND MEMBRANES—

ORMEROD. An Unusual Form of Family Paralysis. *Lancet*, Jan. 2, 1904, p. 17.  
**Meningitis.**—LUNZ. Zwei Fälle von Meningitis basilaris simplex. *Deutsche med. Wchnschr.*, Jan. 7, 1904, S. 61.

STARCK. Hemiplegia superior alternans bei Meningitis tuberculosa. *Beitr. z. Klinik der Tuberkulose*, Bd. 2, H. 1, S. 73.

DUPRÉ, HAUSER et SÉBILLEAU. Méningomyélite tuberculeuse à lésions discrètes, paraplégie aiguë. *Rev. Neurol.*, Déc. 30, 1904, p. 1173.

**Lumbar Puncture.**—CHEVASSE et MAHU. La ponction lombaire dans les complications endocraniennes des otites. *Presse méd.*, T. 11, No. 94, 1903, p. 815.

MACFIE CAMPBELL. Lumbar Puncture. *Rev. Neurol. and Psychiat.*, Jan. 1904, p. 39.

**Polioencephalitis.**—KARA-MURSA. Polioencephalitis superior et inferior chronica. *Inaug.-Dissert.*, Berlin, 1903.

**Encephalomyelitis.**—WILLIAM G. SPILLER. A Case of Intense and General Muscular Atrophy caused by Encephalomyelitis. *Rev. Neurol. and Psychiat.*, Jan. 1904, p. 34.

**Bulbar Paralysis.**—VARIOT. Paralyse partielle de la langue et des lèvres probablement liée à une malformation bulbaire chez une fille de 13 ans. *Bull. de la soc. de pédiat.*, No. 5, 1903.

BONNIER. Un syndrome bulbaire. *Presse méd.*, T. 11, No. 100, 1903, p. 861.

**Intracranial Tumours.**—FRITZ HOPPE. Ein Fall von Psychose in Folge multipler Gehirntumoren. *Neurol. Centralbl.*, Jan. 16, 1904, S. 50.

CESTAN et HALBERSTADT. Epithélioma kystique de l'hypophyse sans hypertrophie du squelette. *Rev. Neurol.*, déc. 30, 1904, p. 1180.

**Cerebral Syphilis.**—TORRL. Intorno a tre casi di sifilide cerebrale. *Clin. mod.*, Dec. 9 e 23, 1903, p. 608.

**General Paralysis.**—MAHAIM. A propos de l'anatomie pathologique de la paralysie générale. *Journ. de Neurol.*, déc. 20, 1903, p. 555.

**Pellagra.**—CARLO CENI e CARLO BESTA. I penicilli nell' etiologia e patogenesi della pellagra. *Riv. Speriment. di Freniatria*, vol. xxix., f. iv., 1903, p. 741.

#### GENERAL AND FUNCTIONAL DISEASES—

**Chorea.**—AUFSCHLAGER. Beitrag zur Localisation der Hemichorea. *Ztschr. f. klin. Med.*, S. 195.

**Epilepsy.**—HENRY LEWIS. An Unusual Epileptic Case. *Brit. Med. Journ.*, Jan. 16, 1904, p. 126.

DECROLY. Troubles dysarthriques permanents chez un enfant pilepti. *Journ. de Neurol.*, Jan. 5, 1904, p. 18.

RANSCHOFF. Hautemphysem nach epileptischen Anfall. *Neurol. Centralbl.*, Jan. 16, 1904, S. 53.

M'COY. Traumatic Epilepsy. *Ann. Surg.*, Part 132, 1903, p. 733.

LUNDBORG. Die progressive Myoklonus-Epilepsie (Unverricht's Myoklonie). Almquist u. Wiksell, Upsala, 1903.

**Neurasthenia.**—PICQUÉ et DAGONET. Sur la neurasthénie post-opératoire. *Méd. scientif.*, Nov. 1903, p. 163.

**Hysteria.**—PAUL SOLLIER. Théorie physiologique de l'hystérie. *Journ. de Neurol.*, Jan. 5, 1904, p. 1.

HELLPACH. Analytische Untersuchungen zur Psychologie der Hysterie. *Centralbl. f. Nervenh. u. Psychiat.*, Dez. 15, 1903, S. 737.

FREDERICK SMITH. Hysterical Paraplegia in the Male. *Brit. Med. Journ.*, Jan. 16, 1904, p. 125.

WESTPHAL. Ein Fall von traumatischer Hysterie mit eigenartigen Dämmerzuständen und dem Symptom des Vorbeiredens. *Deutsche med. Wchnschr.*, Jan. 1, 1904, S. 16.

**Tic.**—LORENZELLI. A propos de quelques cas de tic convulsif. *Gazz. degli ospedali*, Nov. 22, 1904, p. 1478.

- MALHERBE. Tic douloureux facial d'origine syphilitique. *Gaz. méd. de Nantes*, Nov. 14, 1903, p. 984.
- Thomsen's Disease.—JACQUET. Les troubles de la motilité dans la maladie de Thomsen. *Semaine méd.*, vol. 23, No. 47, 1903, p. 381.
- Hydrophobia.—BOSC. Étude et signification des lésions de la rage. Lésions du système nerveux. *Soc. de biol.*, Nov. 13, 1903, p. 1284.
- A. MARIE. Filtrate de substance cérébrale et vaccination antirabique. *Soc. de biol.*, Nov. 13, 1903, p. 1290.
- Acromegaly.—DE BLASIO. Cranio agromegalico. *Riv. mens. de psichiat. forense*, Nov. 1903, p. 487.

#### MENTAL DISEASES—

- G. H. SAVAGE. A Lecture on the Mental Disorders Associated with Decay. *Practitioner*, Dec. 1903, p. 745.
- GAUPP. Zur Frage der kombinierten Psychosen. *Centralbl. f. Nerven- u. Psychiat.*, Dec. 15, 1903, S. 766.
- PAUL JACOBY. Contribution à l'étude de folies dégénératives. *Arch. d'anthrop. crim.*, T. xviii., No. 120, 1903, p. 769.
- PILCZ. Beiträge zur Klinik der periodischen Psychosen. *Monatsschr. f. Psychiat. u. Neurol.*, Bd. 14, H. 6, S. 484.
- JOFFROY. Délires systématisés spirites. *Arch. gén. de méd.*, Jan. 12, 1904, p. 85.
- MICHEL YANNIRIS. L'aliénation mentale en Grèce. Étude statistique. *Ann. méd.-psychol.*, jan.-fév. 1904, p. 60.
- PASTUREL. De l'influence des sorties prématurées dans certains états mélancoliques. *Ann. méd.-psychol.*, jan.-fév. 1904, p. 36.
- WLADIMIR SERBSKY. Contribution à l'étude de la démence précoce. *Ann. méd.-psychol.*, jan.-fév. 1904, p. 19.
- CONOLLY NORMAN. A Clinical Lecture on Hallucination. *Lancet*, Jan. 2, 1904, p. 3.
- BINET SANGLÉ. Le prophète Samuel (suite). *Ann. méd.-psychol.*, jan.-fév. 1904, p. 5.
- DUVIELLA. Sur quelques cas de délire d'origine onirique dans la paralysie générale. Gounouilhou, Bordeaux, 1903.
- DARCANNE. Contribution à l'étude clinique des monologues chez les aliénés. *Arch. de Neurol.*, vol. xvi., No. 96, 1903, p. 479.
- CAPGRAS. Maladies unilatérales de l'oreille avec des hallucinations de l'ouïe (cas de guérison simultanée d'otite externe unilatérale et d'hallucinations auditives homolatérales). *Arch. de Neurol.*, vol. xvi., No. 96, 1903, p. 500.
- SOUZA VALLADARES. L'identification des récidivistes en Portugal. *Arch. d'anthrop. crim.*, T. xviii., No. 120, 1903, p. 806.

#### TREATMENT—

- HOLST. Welche Geisteskranken gehören in die Irren-Anstalt? Jonck & Poliewsky, Riga, 1903, M. 0.40.
- ROBERT JONES. How to Treat a Case of Insanity. *Lancet*, Dec. 26, 1903, p. 1775.

#### ALCOHOLISM—

- G. SIMS WOODHEAD. Recent Researches on the Action of Alcohol in Health and Sickness. *Brit. Journ. of Inebriety*, Jan. 1904, p. 116.
- LANG GORDON. Alcoholism and Heredity. *Brit. Journ. of Inebriety*, Jan. 1904, p. 202.
- GEORGE PERNET. Alcohol and Primitive Man. *Brit. Journ. of Inebriety*, Jan. 1904, p. 210.
- ARCHDALL REID. Human Evolution and Alcohol. *Brit. Journ. of Inebriety*, Jan. 1904, p. 186.
- VOISIN. Inebriety in Jersey. *Brit. Journ. of Inebriety*, Jan. 1904, p. 168.
- G. H. R. DABBS. A Note on the Study of Inebriety: The Unwritten Book. *Brit. Journ. of Inebriety*, Jan. 1904, p. 212.
- W. J. COLLINS. The Institutional Treatment of Inebriety. *Brit. Journ. of Inebriety*, Jan. 1904, p. 97.

## CRANIAL NERVES—

- Facial Paralysis.**—HEWAT FRASER. A Case of Facial Paralysis associated with Herpes Zoster. *Lancet*, Jan. 2, 1904, p. 18.
- KÖRNER. Ueber den Herpes zoster oticus (Herpes an der Ohrmuschel mit Lähmung des Nervus acusticus und des Nervus facialis). *München. med. Wchnschr.*, Jan. 5, 1904, S. 6.
- HELLER. Contribution à l'étude des paralysies faciales congénitales par agénésie du rocher. *Thèse de Paris*, 1903.
- GRIVOT. Contribution à l'étude de la paralysie faciale otitique. *Thèse de Paris*, 1903.
- Pupil.**—FRIEDLAENDER und KEMPNER. Beitrag zur Kenntnis der hemianoptischen Pupillenstarre. *Neurol. Centralbl.*, Jan. 2, 1904, S. 2.

## REFLEXES—

- MITCHELL STEVENS. A Note on the Tendon Reflexes in Uremia. *Brit. Med. Journ.*, Jan. 16, 1904, p. 123.
- F. v. SÖLDER. Ueber den Corneomandibular-reflex. Entgegnung auf J. Kaplan's Einwendungen. *Neurol. Centralbl.*, Jan. 2, 1904, S. 13.
- FUCHS. Ein Reflex im Gesichte. *Neurol. Centralbl.*, Jan. 2, 1904, S. 15.
- MCCARTHY. Ein neues spinomusculäres Phänomen bei normalen Personen. *Neurol. Centralbl.*, Jan. 2, 1904, S. 16.
- TOULOUSE et VURPAS. Étude synthétique des réactions musculaires dans la paralysie générale et essai d'une physiologie générale des réflexes. *Rev. de Psychiat.*, Dec. 1903, p. 502.

## MISCELLANEOUS SYMPTOMS—

- KNAPP. Ein Fall von akut aufgetretener reiner Tastlähmung. *Monatschr. f. Psychiat. u. Neurol.*, Bd. 14, H. 6, 1903, S. 428.
- PEUGNIEZ et PHILIPPE. Hémisection traumatique de la moelle (syndrome de Brown-Séquard). *Arch. de Neurol.*, vol. xvi., No. 96, 1903, p. 486.
- ALTER. Zur Genese einiger Symptome in katatonen Zuständen. *Neurol. Centralbl.*, Jan. 2, 1904, S. 8.
- MANASSE. Ueber erworbenen Hochstand des Schulterblatts. *Berl. Klin. Wchnschr.*, No. 51, 1903.
- BRISSAUD. Les douleurs d'habitude. *Progrès med.*, Jan. 9, 1904, p. 17.
- NAUMANN. Über syphilitische Erkrankung der Wirbelsäule. *Wien. med. Presse*, Jan. 8, 1904, S. 14.

## TREATMENT—

- VAN OORDT. Die Freiluft Liegebehandlung bei Nervosen. *Samml. Klin. Vortr.*, Nr. 364, 1903, S. 77.
- Electrical.**—BORDIER. Résultats fournis par la voltaisation sinusoïdale dans le traitement des névrites. *Arch. d'Électricité méd.*, déc. 15, 1903, p. 713.
- Surgical.**—RAMONÉDE. Médecine opératoire: Résection des nerfs maxillaires supérieur et inférieur à leur sortie du crâne. *Gaz. méd. de Paris*, Jan. 1904, p. 13.
- SABRAZES. L'arrachement des branches du nerf trijumeau dans le traitement de la névralgie trifaciale. *Gaz. hebdom. sciences méd. de Bordeaux*, Oct. 18, 1903, p. 507.
- SCHULZE-BERGE. Ueber Heilung Jackson'schen Epilepsie durch Operation. *Arch. f. klin. Chir.*, Bd. 72, H. 1, 1903, S. 101.
- LUCIEN PICQUÉ. Des rapports de la Chirurgie et de la Psychiatrie. *Rev. de Psychiat.*, Dec. 1903, p. 485.

# Review of Neurology and Psychiatry

## Original Articles

### **SOME UNUSUAL CASES OF TABES DORSALIS WITH REMARKS.**

By ERNEST S. REYNOLDS, M.D. (Lond.), F.R.C.P.,  
Assist. Physician to the Manchester Royal Infirmary ; Visiting Physician  
to the Manchester Workhouse Infirmary.

THE more cases one sees the more one realises that tabes dorsalis frequently shows itself in forms quite different to what one would expect when one remembers the descriptions of the disease so definitely laid down in the older text-books, and the more one is surprised to find when a patient comes complaining of some apparently simple ailment or isolated symptom that one has really to do with a case of tabes. It is not uncommon for patients to present themselves merely complaining of some interference of vision, of an ulcer of the foot, of intractable vomiting, of a swollen joint or of loss of feeling in some part of the body, when on examination an absent knee-jerk and other fairly well-developed and numerous symptoms of tabes are discovered. But if, as in three of the following cases, the knee-jerk is normal, some little difficulty may arise. Moreover, the following cases will show how much better is the term tabes dorsalis than the term locomotor ataxia, for in three of the cases given below there was no ataxia whatever, and in the fourth there was an ataxia in the arms only.

CASE I. Double vision, shooting pains in the legs; knee-jerk normal, no ataxia; later on Argyll-Robertson pupil.

A. B., male, aged 36 years, an engineer, was sent to me by Dr Glascott on 24th February 1899, suffering from paralysis of both vi. cranial nerves, and occasional shooting pains in the legs.

*Previous history.*—About twenty years ago (from date of first visit to me), he thinks he had syphilis; there were sores on the penis, but he had no secondary or tertiary symptoms of any kind. He has been married for several years, his wife has had no children and no miscarriages. Eighteen years ago he had much to do with lead, but apparently has never suffered from lead-poisoning. He takes little or no alcohol. Two and a half years ago he commenced with double vision, due to paralysis of the left vi. cranial nerve, and twelve months ago the pains in the legs commenced; these were shooting in character, each lasting about half a minute, and each attack continuing for about twelve hours.

*Condition on 24th February 1899.*—He has very slight paralysis of both vi. cranial nerves with double vision in the horizontal line. Beyond this and the occasional pains in the legs there are no other symptoms. The knee-jerks are normal, there is no ataxia, no patches of anæsthesia, and no bladder trouble.

He was ordered mercury and potassium iodide, which he continued for one month, and the pains disappeared. He stopped the medicine for three weeks and the pains returned in the left leg, thigh and groin. On 6th November 1899, when he again saw me, he said he had had several severe attacks of pain which I noted as being very like those of tabes; the left pupil now acted very sluggishly to light, and in the centre of each disc the physiological cupping was intensely white, but probably this was of no significance. On 9th May 1900, the paralysis of both sixth cranial nerves had almost disappeared, and both pupils acted well to light. On 14th January 1901, my note reads: "Patient was very well up to four days ago, when he had an attack of pain which came on slowly in the evening and increased the next day. These pains were shooting in character, first on the inside of the knee and afterwards on the outside of the foot, going down to the toes. The paralysis of both sixth nerves has now entirely disappeared."

On 22nd October 1901 the pains in the legs, toes, heels and

knees had increased three days before. They continued for twenty-four hours. The right pupil does not act to light and the left pupil very slightly to light; both act to accommodation. He was again ordered potassium iodide.

July 11th, 1902. He has again had attacks of pain with some numbness on the inside of the right leg, in the groin, and at the end of the penis. Knee-jerks still present and absolutely no ataxia.

February 9th, 1904. Both pupils are small and act to accommodation, the left does not act to light, the right very slightly reacts, neither dilates on sensory stimulation. The knee-jerks are normal, there is no ataxia; the numbness described in the last note has all disappeared. He has occasionally, but seldom, an attack of shooting pains in the legs, which keeps him at home for a day. He takes potassium iodide from time to time, and this he says is the only thing which relieves his pain. Optic discs as before.

**CASE II.** Numbness in distribution of lower sacral roots, Argyll-Robertson pupil, knee-jerks normal, no ataxia.

C. D., male, aged 50, a manufacturer, came to see me on May 11th, 1900, suffering from numbness in the right leg and from much mental distress, as he was afraid of becoming paralysed.

*Previous history.*—He had syphilis nineteen years ago, and was thoroughly treated for this. He has been married fourteen years and has no children, and his wife has had no miscarriages. Some years ago he had slight pain along the course of the right fifth dorsal nerve. About seven weeks ago his feet felt cold, and the toes, first of the right foot and afterwards of the left, became numb; this numbness worked up the right leg, back and front, and afterwards up the back of the right thigh and into the penis, so that he had no erections. It did not spread up the left leg. He has had no shooting pains in the legs.

*Present state* (May 11th, 1900).—There is neither ataxic gait nor static ataxia, the knee-jerks are normal. There is no loss of power. He says he cannot feel anything round the anus, so that he does not know when the motions are passing, but there is no incontinence of fæces, and the bladder acts normally. There is slight but distinct numbness (but no total

anæsthesia or analgesia) in the penis, the perineum, down the back of the right thigh, the front and back of the right leg and the toes of both feet. There is marked myosis of both pupils, but both act very slightly to light, and well to accommodation. There is a very white patch in the centre of each disc, but no interference with vision. He was ordered mercury and potassium iodide.

On May 14th, 1900, he saw me again, and complained that he had had twitching in the legs and arms, and the numbness had extended to a slightly higher level on the sacrum, with slight numbness at times first of one and then of the other inner aspects of the forearms (ulnar nerve distribution). On September 11th, 1900, he was much better, the sensation returning slowly, but the pupils are still small and neither react to light. On November 12th, 1900, I noted that "the right pupil is smaller than the left and acted very faintly to light, the left not contracting to light; both acted to accommodation. There is very slight loss of sensation both to pain and touch in the distribution of the third and fourth sacral nerves; but passage of the motions can now be felt, and sensation has returned in the penis. There is occasionally a slight burning sensation in the right foot." On May 13th, 1901, he had a hypersensitive patch on the outer side of the right foot and slightly of the left foot, and also in the perineum; the pupils are not quite so small, and the right acts more readily to light, the left not at all. On October 16th, 1901, he complained of a sensation across the loins as if a cold wet bandage were applied there. During 1902 he was in every way much better; in fact, so well that, although he had been very nervous about his condition he never consulted a doctor the whole year. He travelled in Italy and Sicily in the winter 1902-03, and kept well until March 1903, when he had a slight return of numbness of the penis. He again was ordered mercury and potassium iodide, which he said always relieved his symptoms. In April he was again better, but at the end of May the coldness of the penis, the hypersensitiveness of the perineum, and the burning of the right foot and leg returned. On June 29th he complained of stiffness and numbness of the right heel, and the burning pain had affected the left foot and also both legs half-way up to the knees. At the end of September he again had some soreness



and pain on the left buttock, with hypersensitiveness across the bottom of the abdomen, and the smarting of the feet and ankles at night had grown worse. I saw him last in January 1904, in consultation with his doctor. He had been in bed for about a fortnight, suffering from influenzal pneumonia of a mild type. His nervous symptoms were better in every way, the only sign remaining being the double myosis and loss of light reflex in the left eye.

**CASE III.** Argyll-Robertson pupil, pains and ataxia in arms only, knee-jerks normal.

E. F., male, age 47, a club steward, was sent to me by Dr Horton-Smith, of Northwich, on August 12th, 1903.

*Previous history.*—He complained that he had been suffering for about five months with occasional tingling and numbness in the hands, especially in the palms. It first occurred in the thumb and first two fingers of the right hand, then in the same positions of the left hand, and now it is also in the ring and little finger of the right hand, but not of the left; he also complains of some shooting pain in both arms. He used to be an engine-driver. He has been married for twenty-two years, has no children, and his wife has had no miscarriages. About twenty-four years ago he had a urethral discharge, and "a lump in the groin which burst." There are no other signs or history of syphilis. He had double vision three years ago, which lasted for three months.

*Present condition* (August 12th, 1903).—Knee-jerks both present, but the left is more marked than the right, the gait is normal, and there is no static ataxia. There is marked ataxia of the hands and arms, which show great awkwardness when he attempts some definite act with the eyes closed, as in buttoning up his coat or touching his nose, this being more marked in the right hand. He has pains in the arms, burning and paræsthesiæ in the hands as above mentioned. His pupils are very contracted, do not act to light, but act well to accommodation. Optic discs normal. He was ordered mercury and potassium iodide.

On October 14th, 1903. He was improving slightly until a week ago, when he noticed that the pains shooting down both ulnar nerve distributions were accompanied by a sudden

contraction of the fingers and of the elbow joints. He says the whole of the right arm feels as if bandaged up in sticking plaister.

January 27th, 1904. He has now great loss of the muscular sense of both hands with general numbness, so that, for instance, he cannot tell without looking whether he has put the whole of his hand into his overcoat pocket or only a few fingers. On testing, the numbness both to touch and pain is only slight; but there is delayed sensation in both hands, but not in the legs. On the whole, the pains in the hands are better; he says he has occasionally had pain across the back of the shoulder; knee-jerks normal, no ataxia of the legs, no bladder symptoms.

CASE IV. Paroxysmal attacks of diarrhoea, then attacks of enteralgia, loss of knee-jerk, Argyll-Robertson pupil; no other symptoms.

G. H., male, aged 66, an estate agent, came to see me on June 2nd, 1903, complaining of sudden and repeated attacks of diarrhoea.

*Previous history.*—He has never had syphilis, so far as he knows, but had one or two attacks of gonorrhoea before he was thirty years of age. He has been married twice, has three children by the first wife and two by the second, all healthy, no miscarriages. He told me that he commenced with the attacks of diarrhoea on January 24th, 1902, and that they continued for some months; they generally came on during the night, and, as a rule, were better in the daytime. They were preceded by slight colic or by tenesmus. They were better if he was away from home. Five weeks ago the attacks started again and continued up to his visit to me.

*Present State (June 2nd, 1903).*—The patient is short in stature, with very square shoulders, and no apparent deformity. There was nothing to be found abnormal in the abdomen, rectum, or in the urine. I found on inquiry that he had very great home worries, and he accepted readily my idea that his attacks were nervous in origin and set up by his worries. I ordered him to go from home, and gave him some simple astringent medicine.

On February 2nd, 1904, he called again to see me, saying that the diarrhoea was much better, and only occurred occa-

sionally after much exertion. But during the last week, after any exertion, such as gentle walking, he has had severe pain in the lower part of the abdomen, which only passes off after resting. This was not followed by diarrhoea, and was, indeed, a new kind of pain. His description was something like the description of a girdle pain, and on examining his back I found a prominence of the tenth and eleventh dorsal vertebræ, but he said that had been there since childhood, and that it never troubled him; in fact, he hardly seemed to think it was abnormal. But as I wondered whether he was developing some pressure on the cord I examined his knee-jerks, and found to my surprise that they were entirely absent. There was no atrophy of muscle, however, and no bladder symptoms, so that it was unlikely the absent knee-jerks were caused by pressure on the lumbar enlargement. I then examined his pupils, and found both myotic, left slightly larger than right, neither acting to light, but both to accommodation; the discs were normal. There was no trace of any form of ataxia. Inquiry elicited the fact that for some years he has had occasional shooting pains in the legs (not the joints), which he ascribes to rheumatism, and which he says are sometimes accompanied by burning and scalding in the feet and legs. He has had no bladder trouble.

#### COMMENTARY.

There are some points of interest in these cases which I should like to mention.

*Diagnosis.*—As regards the diagnosis, I think there can be no doubt that the first three patients were suffering from tabes dorsalis, in spite of the fact that in all the knee-jerks were normal, and in the first two there was no ataxia. In the third case, apparently, there is a pure cervical tabes, which is certainly rare; it is the first case of the kind, so far as I remember, which I have personally met with in over twenty-five years' experience. The fourth case is, I think, a doubtful case of tabes, but it seems to me to be the only diagnosis which will explain all the symptoms. It is particularly interesting, as there was nothing at first sight either in the history or the appearance of the patient which would have suggested that the diarrhoea was really that found so rarely in tabes, and it was only the sus-

picion that his later pains were something in the nature of a girdle pain which led me to examine the knee-jerks and the pupils.

*Argyll-Robertson Pupil.*—All the cases showed this sign, certainly one of the most important clinical signs to be investigated in nervous disease. There seems to be still much doubt as to the exact position of the lesion which causes the loss of light reflex so often combined with myosis and loss of the dilatation reflex from sensory stimulation. Possibly, just as in the cord, there is a selective action by the disease agent, there is also a selective action on the dilator and light reflex motor nuclei in the upper part of the pons; and similarly, in the early stages of tabes (but very rarely in general paralysis), a selective affection of the nerve nuclei supplying the muscles external to the eyeball, causing paralysis so often temporary in character. It will be noted that in Cases I. and II. the light reflexes have varied from time to time in a very curious way, sometimes being present only in one eye, sometimes only in the other.

As regards the clinical significance of the Argyll-Robertson pupil, I cannot remember any cases apart from tabes dorsalis or general paralysis of the insane in which it was manifested; and I certainly agree with Dr Mitchell Clarke's statement (*Brit. Med. Journ.*, Jan. 1904) that it is not present in any syphilitic nervous disease apart from general paralysis or tabes, and therefore if it is a post-syphilitic affection at all, it must be in the nature of a para-syphilitic change, or at any rate is only produced by the same pathological cause which sets up the chronic inflammatory changes of the posterior columns or of the brain cortex. For in my opinion it is by no means proved that every case of general paralysis and tabes is due to syphilis. It is, for instance, not at all an uncommon experience for me to obtain a definite history of gonorrhœa in tabetic cases, but to absolutely fail to get any history or evidence of syphilis. Now it certainly seems to me more or less unscientific, and a closing of the door to future enquiry, to assume because a person gets such a common affection as gonorrhœa and subsequently shows signs of tabes, that his gonorrhœa must have been accompanied by syphilis in spite of all evidence to the contrary.

A point which has often struck me, and which is noticeable in the first three of my cases, is that not unfrequently tabetic

patients are childless, apart altogether from miscarriages. It would be interesting to know whether this sterility should be taken as indicating previous syphilis.

Other apparently slight signs which were of importance in diagnosing the cases given were previous double vision, which occurred in two of the cases, and implication of sensory nerve roots, as shown by anæsthesia or paræsthesia, along certain definite tracts, such as occurred in the second and third cases. Such limited sensory phenomena should always lead to a thorough examination for tabes.

The first two cases show, I think, the good effects of anti-syphilitic treatment in the early stages. In spite of my remarks above, I regard syphilis as the most important predisposing cause of tabes in probably 90 per cent. of the cases, and for this reason in the early stages of the affection I always order the patient mercury and the iodides with, I believe, good results. Taking the first two cases as examples, I feel certain, though it is difficult or impossible to prove, that the energetic anti-syphilitic treatment, renewed from time to time, has been the means of converting what would have otherwise been a steady progress of the disease into a slight recovery, or at any rate an arrest in the symptoms.

When the disease is well established, I have never seen anti-syphilitic treatment do the slightest good.

## **A MORPHOLOGICAL CONTINUITY OF GERM-CELLS AS THE BASIS OF HEREDITY AND VARIATION.**

By J. BEARD, D.Sc.,

University Lecturer in Comparative Embryology, Edinburgh.

*(Continued from p. 142.)*

### **IX. THE CAUSE OF GENETIC VARIATION.**

THE Darwinian theory starts from the existence and facts of variation,<sup>1</sup> and, regarding these in the light of natural selection and the struggle for existence, finally concludes that these factors result in a survival of the fittest. From a general review of the

<sup>1</sup> "The foundation of the Darwinian theory is the variability of species."—*Wallace*.

theory of natural selection from the point of view of embryology, one or two things are clear. Natural selection and the struggle for existence can only be really effective if the individuals, i.e. the big masses of such, experience all the influences of the struggle and selection before they breed. But, as every physician knows, no struggle for existence and no natural selection are powerful enough at the present time to weed out anything like all the weaklings, mental and physical, before the period of sexual maturity. And some types of individuals, characterised by marked mental or physical degeneration, exhibit little or no effects of natural selection in retarding or diminishing their fecundity; in fact, the actual conditions observed are frequently the very opposite. Without doubt there are other factors far more potent for the elimination of organisms than natural selection. An instance may be given. In man the normal proportion of the sexes at birth is 106 males:100 females. Between this period and sexual maturity this alters, and for England and Wales it is in young adults (of 20-25 years) 91 males:100 females. Natural selection and the struggle for existence may, if one will, be taken as accounting for this latter difference. But, as Rauber and von Lenhossék have shown, very different figures are obtained if abortions be taken account of.

From the examination of 57 human *fœtuses*, Rauber (16) found the proportion of the sexes to be 159:100, while von Lenhossék (17), with a much greater material of human *fœtuses* of the third to the sixth month at his disposal, found among 156 specimens 96 males and 60 females. If the results of the two observers be taken together, the proportion works out to about 160 males to 100 females.

Again, if one take account of still-born children only, the average proportion for the chief races of Europe is about 132:100. Not before the third month of gestation is it possible from a superficial examination of the human *fœtus* to determine its sex. It is probably for this reason that no similar observations upon abortions of the first two months have been carried out. But, as above shown, the proportion of males rises from 106 through 132 to 160:100, and probably, could one take a census of all the eggs fertilised, it would be found that for every female egg fertilised there were two male ones, or that the real proportion

would be, not that at birth, viz., 106:100, but 200:100—2:1! This is explained by the diagram of oogenesis (p. 122, Fig. 8), for it has been concluded, for other reasons, that one main difference between the male and the female egg, of the skate and probably of man also, is to be found in the occurrence of an additional division in the oogonia before they become oocytes.

There can be no doubt that a basis of truth underlies the foregoing statement; and it would follow that nearly half of the male individuals of mankind were eliminated before the birth-period was reached! Why this happens can only be surmised. Some embryologists maintain the action of natural selection upon the embryo or foetus *in utero*, but this statement rests on no evidence. Possibly the process is a result of the swinging and swaying of the self-regulating mechanism of sex described elsewhere.

The term "*amphimixis*," to denote the mingling ("*Vermischung*") of two "*Keimplasmas*," was introduced some years ago by Weismann. Even in some parts of his latest work (e.g. p. 216, vol. ii.) it is still used in this sense. According to him, the hereditary tendencies of two individuals are united (by the junction of egg and sperm) together, "and the organism, whose formation is derived from this mixed germ-plasm, must, therefore, take on characters from both parental individuals, in a sense be made up of traits (*Züge*) of both parents."<sup>1</sup> This is the one result of *amphimixis*, whilst the other, brought about by the (under Weismann's views) complicated machinery of the reducing division, which results in new combinations of the "ids" or groups of characters, is the preservation of individual differences by means of the continually fresh combinations of characters already present in the species.

In this way all the phenomena embraced by Weismann in the conception of *amphimixis* become a cause of variation, but in his words, "not the real root of variation itself." As will appear later, the actual cause of variation is referred by

<sup>1</sup> Under the views to be advanced here this must be disputed. Owing to the union of an egg and a sperm, the resulting individual *may therefore*, but not *must therefore*, be made up of characters of both parents. It is also denied that *amphimixis*, i.e. the union of egg and sperm, is to be regarded as a "mingling," and in an earlier chapter the existence of the so-called reducing division has already been denied, and with it the complicated machinery of the "new combinations of ids," etc.

Weismann to certain complicated phenomena, termed by him "germinal selection."

The term "*amphimixis*" is undoubtedly a convenient one, and it would be still more useful, were its meaning not conditioned to so great a degree by the hypotheses of Weismann and his pupils relating to the "reducing division." For this and other reasons to be presently mentioned, the term will not be adopted in the present writing. Even though the existence of the germ-plasm were admitted, the known facts do not point to the union of egg and sperm as the mingling of two such germ-plasmas, or as a mixing of two individualities. This must be strongly insisted upon as against the views of Weismann and Ewart. Owing to the nuclear duplication (Figs. 1 and 2), observable from the time of fertilisation to much later periods of the cycle, fertilisation is not in any sense *the mingling* of two germ-plasmas (Weismann), or even of two lines of ancestry (Ewart and others), but it is merely the joining-together of two (potential) sets of characters, of two individualities. And again, it is not clear that the subsequently formed offspring must therefore take on characters from *both* parents, for known facts go to show that it might be made up almost or even entirely of characters belonging to the line of one parent only, or, apparently, even to neither. Moreover, how can Weismann bring the Mendelian results, to be afterwards described, into line with his views? In his latest work (vol. ii. p. 64) Mendel's experiments are, indeed, mentioned, but no attempt is made to account for their results.

The definition of *amphimixis*, given by Weismann in his earlier writings and referred to above, receives still further additions on p. 297, vol. ii. of his recent work, where he writes: "To-day *amphimixis* in the entire world of organisms, from the unicellular ones up to the highest plants and animals, has the meaning ('Bedeutung') of an increase in the capacity for adaptation of organisms to their surroundings, in that by it the simultaneous harmonic adaptation of many parts is possible. It accomplishes this by the continual new combination of the germ-plasmids of different individuals, and thus furnishes the processes of selection with the means of fostering the favourable ones, and of eliminating the unfavourable tendencies to variation, as well as the collection and union of all the variations



necessary for the proper further development of a species. *This indirect action of amphimixis upon the preservation and transformation-powers of living forms is the chief reason of its general introduction and retention in the whole known kingdom of organisms from the unicellular ones upwards.*"<sup>1</sup>

A clear conception should be formed of all that Weismann includes under *amphimixis*. The new combinations of chromosomes, of groups of characters, which under his views happen at the reducing division—the last of the two, universally assumed<sup>2</sup> for the Metazoa by him and his followers, and which precedes the actual formation of gametes—lead to what one might almost describe as an infinite, certainly a very great variety of, gametes, and this would of necessity be such, that of the four sperms formed by a spermatocyte in, say, a fish, no two could be alike in their characters. Indeed, if this be correct, it is not the *differences* among the progeny of any two parents which require explanation—the phenomena of the reduction amply accounting for these—but the *resemblances* to one another and to their parents. It is, therefore, under Weismann's views, the initial differences in the gametes, which in the new combinations of the germ-plasms really furnish such abundant material for the continued action of germinal selection (in the sense of Weismann), the *mingling* of the qualities of the germ-plasms of the two gametes determining the organism.<sup>3</sup>

Anticipating what is to follow, let it be here stated with emphasis that those theories of germinal selection or variation which, like Weismann's and Ewart's, advocate an elimination of individuals (natural selection) and the reduction of chromosomes as happening at one or other of the usual two final divisions prior to the formation of gametes, cannot have any identity with the conclusions to be presently set forth. And with this statement a reference may be made to the germinal selection of

<sup>1</sup> This passage may suffice to demonstrate the absence of correspondence between Weismann's views of the cause of variation and mine. As will appear later, where Weismann sees but chance, there certainty may be recognised.

<sup>2</sup> Be it remembered, that in no mammal is more than one polar body known, and that, if Moore's researches be correct—and I for one do not doubt them—here also the spermatocytes divide once only. These facts have not as yet been impugned.

<sup>3</sup> Under Weismann's views it is, of course, germinal selection which furnishes the material for natural selection to work upon.

Weismann. While he has urged the "all-sufficiency" of natural selection against the late Herbert Spencer, he has reinforced it very considerably by the conception of a germinal selection. It is difficult in the absence of any authoritative definition of the process to describe what Weismann understands under germinal selection. It is purely a mental concept, and it is not brought into definite connection with any known epoch or phenomenon of the development. *It hangs entirely in the air!* Underlying it is the assumption (vol. ii. p. 141) that the determinants, those entities which decide the characters "of every part of the organism, must be contained several times or many times within it; while, on the other hand, every 'id,' made up of determinants, contains potentially the whole organism." What a terrific multiplication of causes! The child is therefore not determined by the determinants of one id, but by those of many ids, and the variations of any part of the body depend not on the alteration of a single determinant X, but on the combined workings of all the determinants X, as they are contained in the total ids of the particular germ-plasm. The prime cause of the individual variation is set down to passive accidental (!) oscillations in nutrition (p. 138). Most remarkable of all, he writes that germinal selection is quite independent of the environment!<sup>1</sup>

In this respect Weismann differs from Ewart; the latter speaking not only of a germinal variation, but also of an environmental one. "As some variations which might very well be looked upon as acquired (*e.g.* variations due to differences in age and vigour, and to ripeness of germ-cells) are transmitted, I propose speaking of variations as germinal and environmental. The most critical and momentous period in the life-history of any plant or animal is during the conjugation of the male and female germ-cells. During conjugation (fertilisation), as the germ-cells more or less completely blend with each other, and as new combinations, partly chemical and partly mechanical, are rapidly formed, the fate of the new individual is largely fixed. It is not

<sup>1</sup> Weismann, A., "Vorträge, etc.," vol. ii. p. 134, 135. As it will be maintained that both variation and selection depend entirely upon the interactions between organisms (the germ-cells) and the environment, the actual passage may be cited in full. "Darin liegt gerade die hohe Bedeutung dieses Kräftespiels im Keimplasma, dass es ganz unabhängig von den Beziehungen des Organismus zur Aussenwelt Variationen schafft."

so much that the conjugation causes variation, as that effect is given to variations inherited from near and remote ancestors, or accumulated during the growth and maturation of the germ-cells. It is to this variation, which inevitably flows from the blending of two highly specialised germ-cells, I have given the name *Germinal Variation*. During conjugation the minute details of the new individual may not be settled, but undoubtedly more occurs than the laying of the foundations. Subsequent to conjugation there is considerable scope for variation in the size, colour, vigour, etc., of the new individual, as there are possibilities of various changes of the germ-cells prior to conjugation. All the variations in the germ-cells up to the moment of conjugation together with the variations during development and growth, I shall refer to as *Environmental Variation*" (18).

It is not proposed, and it is impossible in the space at disposal, to give adequate accounts of the views held by other investigators as to the cause or causes of variation; and the above citations are made only to demonstrate to the reader that the conclusions to be presently advanced are as novel as they are simple and sufficient. As recently, indeed, as 1902, Bateson wrote: "In other words, it is *the cause of variation* we are here seeking. To attack that problem no one has as yet shown the way. Knowledge of a different order is wanted for that task; and a compilation of ancestry, valuable as the exercise may be, does not provide that particular kind of knowledge" (19). Certainly, it does not! Were the solution of the cause of variation to be found in an enforced and illegitimate union of higher mathematics with the sciences of living things, and the study therewith of non-existent "ancestors," it would probably have found an easier and simpler explanation long ago in some or other of the laborious investigations of Darwin, for instance, in the results revealed by artificial selection.

The problem of the cause of variation, like many other obscure riddles of science, belongs to that branch of human knowledge termed embryology. Surely, one day mankind will awaken to a recognition of the possible existence and to the overwhelming import of such a science, and as certainly *then* it will be seen, that all problems relating in any way to reproduction are ultimately questions of embryology! If a child be not the exact image of either of its parents, it is in embryology that the

solution must be sought. If there be varieties in a species, if new varieties appear, if species be discontinuous, it is ultimately embryology which must offer the true explanations. It is many years since the writer first read "The Origin of Species": it has since been studied many times. But one of the impressions then gathered from that great work is still vivid, viz., the very minor part which embryology really plays in Darwin's investigations.<sup>1</sup> Of later workers Weismann has possibly assigned the greatest importance to the facts and methods of embryology, but with the utmost respect it may be submitted, that many of his embryological views are more theoretically embryological than actually, and, as other portions of the present writing reveal, there is an impassable gulf fixed between the embryology of Weismann and the conceptions of that science, *advocated as the results of investigation*, by the writer.<sup>2</sup>

With the latter an approach of the problem of genetic variation is now made for the first time, and at this stage it may be well to recall the farthest point, leading anywhere in this direction, which his published investigations had yet reached. In a recent memoir on "The Determination of Sex" (p. 756), the following passages occur:—

"1. The union of the nuclei of the two gametes in fertilisation is the joining of two individualities. In Haecker's terminology their autonomy is retained along the germinal track, and in the primary germ-cells.

2. In the primary germ-cell, which unfolds itself as an embryo, this autonomy is shattered, with the resulting conquest of the stronger, i.e. more potent portion. As each nucleus is made up of a series of characters or qualities, this conquest may be on the part of all those of one nuclear half, or of the greater or less number than half of those of either portion. That is to say, the offspring will reflect the sum-total of the half of all the

<sup>1</sup> Nor need this be wondered at. In Darwin's student-days there was no possibility of his obtaining any knowledge of embryology at either Edinburgh or Cambridge. The then Professor of Natural History in Edinburgh was a mineralogist! Even now things have not greatly improved, and as yet there is not a single Chair of Embryology in Great Britain.

<sup>2</sup> In this connection a comparison of Fig. 5 of the present writing, showing the *whole* track of germinal continuity from generation to generation, with Weismann's or Boveri's figures of the germ-track of *Ascaris*—for *five cleavages only*!—should be made. No germinal continuity is shown in the latter, for the further fate of all the cells is unknown!

characters of the two lines, paternal and maternal, represented each by a unit in each of the gametes. For clearness, if all the paternal characters contained in the sperm, and, therefore, in the spermatie half of each nucleus along the germinal track, be represented by a red pack of cards, and if all the maternal characters of the egg be symbolised by a blue pack of cards, the offspring will be made up of characters which together make up a complete pack of cards (not two such), red or blue in any proportion. The duplication in the nuclear elements is equivalent to a *doubling of all the characters handed down along the two lines of the egg and sperm. The 'embryo' can only contain half of these characters.*

3. "As already indicated, the reduction of chromosomes must originally have been merely an undoing of the previous conjugation, such that each separated half became a gamete, or gave rise to such. This separation originally took place at the division of the primary germ-cells into secondary ones. By the intercalation of new mitoses it has been delayed until the end of this intercalated series, until the division of the oogonia into oocytes, for example. The result of this has been, that the reduction is no longer effected by cell-division, but it appears in the closing phases of such a division.

4. "The one half of each nucleus, which originally, like the rest, itself gave rise to gametes, owing to the separation (Figs. 8 and 9), due to the intercalation of new mitoses, is thus lost in and during the reduction.<sup>1</sup> The separation along two lines to form gametes having taken place at an earlier point, it cannot be repeated.

"In considering the reduction and allied questions, such as those of heredity, two factors must not be overlooked. These are, that, on the one hand, the line of 'ancestry' ends in the 'embryo,' which forms a termination of two lines of 'ancestors,' paternal and maternal, and that, on the other, it is continued onwards for the germ-cells *without passing through the embryo*. This has hitherto, owing to the nature of prevailing

<sup>1</sup> In the following more light will be thrown upon the true meaning of the reduction, and it will become apparent why it is not effected by mitosis. The reduction of chromosomes is certainly an undoing of the previous union, but much more than this, and there would appear to be no reason for supposing that it was ever effected by cell-division. In the following the statements made in paragraphs 3 and 4 will be very considerably altered.

conceptions of the relationships of embryo and germ-cells, been ignored."

And on p. 748 is written: "Not only, therefore, is the period of the reduction of chromosomes associated with that of the determination of sex, but the said reduction is immediately, in both animals and plants, followed by direct manifestations of sex, in the one instance the gametes, in the other the gametophyte. The two are thus linked together. . . . The only logical conclusion would appear to be that the reduction is the outward manifestation of the determination of sex. *Whether the latter be brought to pass by the reduction of chromosomes, or, as is quite possible, the reduction in the number be a consequence of the determination of sex; which of these be the cause and which the effect is a question for research.*"

When writing the foregoing, it was clear to the writer that something else was involved in the disappearance and loss of half of the chromosomes at the period when, for instance, the oogonia become oocytes. The disappearance of half of the chromosomes, like the disappearance or latency of half the characters (contained in the chromosomes), and, therefore, the loss or latency of half the characters of the embryonic cell, seemed to be a remarkable phenomenon with some still deeper-lying meaning.

The following-up of the thread anew leads to the discovery of a simple cause of variation, so elementary in nature, that a simpler one is inconceivable, albeit so potent in its workings that in the explanations it offers of phenomena, and in the mode in which it excludes other causes, it must be *the cause of variation*.

It will be assumed with de Vries and Weismann that there are such things as pangenes or biophores as the ultimate entities of living matter. These biophores, whether arranged in groups as determinants or what, are disposed and contained in the gametes, eggs and sperms, in such a way that in each egg or sperm they represent, like a complete pack of cards, *one and one only* complete set of the *potential* characters or qualities necessary to form an individual of the species. They are contained in the nucleus. Clearly, there is no new conception here. Not only the writer, but Yves Delage, Boveri, and others have recognised this. At fertilisation, therefore, we have, as already seen, the joining, *loose*

*and without blending*, of two individualities, like two packs of cards.<sup>1</sup> In every division up to the formation of the primary germ-cells, a duplication of these characters or qualities is retained, such that each of the daughter-cells contains the double set, as evidenced by the duplicated nuclei. There will thus be in every cell *two representatives* of every character or quality. It is inconceivable that in ordinary sexual reproduction the characters thus duplicated should be identical, though often equivalent. If this be not obvious *a priori*, it will be seen to follow from the subsequent facts. Taking any two characters A and a, or any two cards of the packs, the red nine of diamonds and the blue nine, these will differ in constitution, in size, in every characteristic, if only because derived from two different parents. Both may be equally suitable for the environment, and the consequences of this will appear later. Or again, one, say, the red nine of diamonds, may be better adapted for the environment than the other, the blue nine of diamonds, and so with all the other characters. Anon, comes the epoch of the formation of the primary germ-cells, and one of these draws the lot of developing. If all the characters of both packs be equally adapted to the environment, the cards of the pack chosen to develop will happen according to the mathematical laws of probability, and a pack made up of any combination of red and blue cards will be taken to form the characters of the embryo. If, on the other hand, as is usually far more probable, the cards of the two packs differ in suitability, the better cards will be chosen, and the others will either remain latent or be eliminated.<sup>2</sup>

So much will be clear, but it will also be apparent, that as yet we are no nearer the explanation of the cause of variation. All that subsequently happens in the case of the "embryo" or individual relates to it alone and its reactions to the environment (somatic variation). *The true cause of genetic variation lies in the germ-cells and in the phenomena connected with these.* One might speak of a germinal selection, but

<sup>1</sup> On the other hand, Sedgwick (*Rep. Brit. Assoc.*, 1899, p. 761) says: "The *zygote*, as the mass formed of the fused gametes is called, is formed by the combination of two individualities, and is therefore essentially a new individuality." Man and wife are sometimes spoken of as one, but in other cases  $1+1=2$ !

<sup>2</sup> The fact that the embryonic cells retain the duplication of chromosomes would appear to show that they remain more or less latent. This retention may be of physiological import for the life of the cell, *i.e.* balance and compensation.

it would not be at all in the sense of Weismann's conceptions of this. In the union of two gametes, an egg and a sperm, the joining-together of two individualities, of two complete sets of potential characters, or qualities, as these are of different origin, though often equivalent, they cannot be identical. The two gametes are like two differently coloured packs of uneven cards, such that no two corresponding cards are exactly alike. Such double packs are present in all the germ-cells, which come to lie in the germinal nidus of any embryo. Some writers suppose them to be uninfluenced (!) in any way by their surroundings, and they assert that no matter what the individual containing them may do, this cannot in any way affect the germ-cells! But all living matter must exhibit irritability, feed, and grow; these are the commonplaces of physiology. In fine, the germ-cell must react to and be influenced by its environment. Given an environment of a certain kind, there will be no element of chance in its influence on the germ-cells. But, as the two packs of characters, making up each germ-cell, are not identical, this influence of the environment, including in this everything which can influence the individual and its germ-cells, food, climate, toxins, disease, natural phenomena of all sorts, will differently affect the various corresponding characters of any germ-cell. Some characters will be more favourably or less unfavourably influenced than other corresponding ones, and the former will "grow" and increase in importance, while the latter will be retarded or diminished. In this way, if the environment have not been a constant one, by the time the reduction of chromosomes comes round, the "cards" of the two packs of corresponding "packs" of characters, reacting to the environment, will have altered more or less. At the reduction of chromosomes, happening in the closing phases of the last division of the oogonia into oocytes, and of the spermatogonia into spermatocytes, from the two "packs of characters" a new complete pack will be chosen, and this will include all the favourable characters, the other corresponding ones being excluded. In this way, at the reduction there must happen the elimination of one complete set or characters. According to circumstances the process of selection of the new pack will be such as to include either the whole of one or other of the original packs, or only certain portions of either. The direct forerunners of the gametes, the oocytes and



spermatocytes resulting, are made up, like the original gametes, of one complete set of characters, and this composition is handed on to each and every gamete derived from any one oocyte or spermatocyte. This new pack may, according to the previous action of the environment, possess any possible complexion, from that of including the whole of the characters of one or other of the original gametes, to that of excluding any number of the characters of either of the two, the ones excluded being represented by the corresponding characters of the other pack.

Regarded in this light, the reduction of chromosomes acquires a new significance. With it, as we have seen, is bound up the determination of sex; but as this only relates to the morphological nature of the gametes, *the true meaning of the reduction is the diminution of one set of characters, such that, if among those of the two original sets there be any unsuitable ones, these are rejected.* As there are always two complete sets of characters in the germ-cells prior to the reduction, when this takes place, *the result is the elimination of one complete individuality.* This may sometimes be one of the original ones, with which the union began, when it will really be—a thing of great importance—the elimination of an “ancestor”! Or, if it be only the exclusion of certain different portions of each of the original sets, none the less, it is in effect the same thing as the elimination of one complete individuality.

Moreover, as will appear more clearly subsequently, owing to the up and down oscillations of the characters of the two packs, during their union and under the influence of the environment, and owing to the results given above, the process just briefly described becomes, not only a potent cause of variation, but a *self-regulating mechanism*, by means of which any variety of a species must adapt itself to its environment. And, as will be seen, if the environment change to a considerable extent, it will become the actual cause of the origin of species. Finally, before entering further into the consideration of these matters, it may be insisted that the process described in reality brings about an elimination of individualities, of potential individuals, beside which natural selection is mere child's-play, that it explains all the facts in such a way as to render any other and more complicated construction superfluous, and that it ought to reconcile the views of the Darwinians, on the one side, with those of the followers of Lamarck, on the other.

In the foregoing, as in other inquiries, the writer has endeavoured, in the words of Yves Delage, to limit the hypotheses, and in making one, to keep always in view the point of departure, never the final purpose (20). With the duplicated nucleus and the conception of the gametes, egg and sperm, as complete cells or entities, and not half entities requiring the other as a complement, we are dealing with actual facts. These are not new conceptions, for nowadays they would willingly be subscribed to by many embryologists and botanists, thus by Delage, Boveri, and Strasburger. Many, too, would accede to the view that the egg and sperm each contained all the characters or qualities necessary for forming a new individual, Metazoan or Metaphyte. It is also in accordance with the facts to maintain that the *amphimixis* of Weismann has no real existence; on the contrary, that the union of egg and sperm at fertilisation is merely the joining together in a loose way of two individualities, of two complete sets of characters or qualities.<sup>1</sup> This union is in animals retained by the germ-cells, until the period of the reduction, by the developing embryonic cell until the commencement of the development, when it would appear to become latent, and in plants during the whole life-period of the sporophyte or flowering plant. The two sets of characters thus joined together cannot be identical at the start. As living entities they must, like all living things, react to their surroundings; in a word, they must be influenced by the total environment, nutrition, climate, disease, toxins, etc., etc. The effect of all the factors will be a different one on the differently constituted characters. Some will be favoured by it, and they will flourish and increase in importance; others will be neglected or unfavourably influenced, and these will diminish. When the period of the so-called reduction arrives, there will be a settling-up, and, supposing the environment not to have been a constant one, some characters will be chosen for the new pack, while other corresponding ones, the less favourable ones, will be rejected. In this way an elimination of unsuitable characters will be brought to pass, and this, on occasion, may be such that it might more fitly be termed an elimination of individualities.

<sup>1</sup> A genetic character or quality in a germ-cell is, as Sedgwick remarks, not the character itself, but the possibility (or *potentiality*) of producing it.

And this is the same thing as an elimination of so-called "ancestors."

And, finally, owing to the facts being as stated; because each egg or sperm is a complete individuality, a complete set of characters, because in the union of these we have the joining together of two complete but not identical sets of characters, and because for a greater or less period of time these are subjected to and react to the influences of the environment, etc., *the process becomes a self-regulating mechanism, the up and down oscillations of the characters or qualities of the two sets endeavouring to follow and compensate the changes in the environment, and the result must be genetic variation.*

The process described must, in fact, be a very potent cause of variation, because ever ready to come into action, and always impartially and unmercifully eliminating the unfit. But it is something more than a cause of variation and of varieties; in the long run and on occasion it becomes the cause of the origin of species. In contra-distinction to any other existing complicated theory of germinal selection or variation, it may be defined as *germinal election and elimination, in adaptation to the environment.*

#### X. GERMINAL ELECTION AND ELIMINATION.

One impression gathered from a study of the works of, for instance, Darwin, Wallace, or Weismann, is the immense importance attached to the analogy of artificial selection. It would be wrong, and it would be only an assumption, to imagine that Nature in her workings must of necessity act as does man in artificial selection. Groping blindly in the dark, he picks out individuals possessing certain characters, and eliminates others, in which they are, *apparently*, absent. The latter are in his eyes the unfit or the less fit. What on a small scale he attempts, that—so it is, but wrongly, concluded—Nature does on a large one. Under the analogy of artificial selection she is supposed by natural selection, resulting from the struggle for existence in the survival of the fittest, to eliminate all the unsuitable individuals, and thereby to select those for the continuance of the race which are most or more suitable for the environment. Even though she did this, the result would be as nothing compared with that

elimination of unsuitable characters or qualities, which at its basis is also a weeding-out of individualities, and which under sexual reproduction she obtains in the duplication of every character or quality. A selection of individual organisms, though they be the bearers of germ-cells, which in the meantime are so many understudies of the individuals themselves, can give no certain result, either for artificial or for natural selection. For in those germ-cells at the reduction an election of certain characters and an elimination of others have still to be made, and, moreover, a new union with an unknown quantity, another germ-cell, has to be entered upon. In artificial selection man does not wittingly, as a rule, even call in the aid of the decisive factor, the environment!

Nature goes to the root of the matter: she makes no selection of individuals, for about these she cares nothing.

"Are God and Nature then at strife,  
That Nature lends such evil dreams?  
So careful of the type she seems,  
So careless of the single life:"—TENNYSON.

She can exercise her choice, and she does it, among the germ-cells, and not merely in these, but among the characters or qualities the germ-cells possess. In this it would be futile to attempt to bind her down by cast-iron laws of inheritance, to dictate that "the average contribution" of a father should be so much, of a grandfather so much, and so on. This may hold good in cases, but only with a constant environment. When the latter obtain, as will be seen, if all the characters or qualities be equally good, the election at the reduction may be left to the mathematical laws of probability; they may be taken apparently at random, and in this way it may become possible to speak of sexual reproduction as *sometimes* a mingling or *amphimixis* of characters, and to set up laws of inheritance by average contribution.

According to Weismann, even Huxley would appear to have doubted the adequacy of natural selection. For Weismann himself it is "all-sufficient"; none the less, has he not set up "germinal selection" to reinforce it, and to furnish the material for its action? Under natural selection one little thing, any trifle, may decide life or death, and the individuals of a

species become of the utmost importance as factors. In the whole doctrine one may read the application of artificial selection, as exercised by man, to a larger area. But it is, for our purpose, not needful to inquire into the justification for a belief in natural selection. Granting that it exist, and that in its workings it be only similar, but on a vastly greater scale, to what man can effect in artificial selection; what it would do, that, *and far more than that*, Nature brings about in a more efficient way by selecting in the germ-cell which of two characters or qualities, the greater or the less, shall be taken. Man works by haphazard, and were one to take into account the number of times in which in his writings Weismann attributes particular things to "chance,"<sup>1</sup> it might be concluded that Nature did the same. But in her selections the element of chance is absent.

With a constant environment, or with what is assumed to be such, man first eliminates, *i.e.* rejects (individuals of) certain varieties, and in this way favours (individuals of) some particular variety. By closely intercrossing these, he accentuates particular points, because, of course, even in the characters of germ-cells suited to an environment there may be degrees. In artificial selection (elimination of Lloyd Morgan), man takes a course the reverse of that adopted by Nature. Her method may be slower, *but it is sure!* When she causes variation, she initiates it by altering the environment, and while some one or more varieties of a species may be able to adapt themselves to the new environment, others will fail in this. These latter will, of course, at once disappear, if they be not fertile with the more suitable varieties. But one need not, as Romanes did, make any assumption of infertility. The environment has been taken to have been rendered suitable for one or a less number than before of existing varieties. By reproduction these other varieties, so far as they are fertile with the suitable variety or varieties, will then be eliminated by the self-regulating mechanism, and new ones will arise under the same self-regulating mechanism of the germ-cells and environment, from that or those suitable for the environment.

It would most unduly lengthen out the present writing were an attempt made to cover the whole field of the observed pheno-

<sup>1</sup> Even in germinal selection. Compare "Vorträge, etc.," vol. ii. pp. 141, 149, and 369.

mena.<sup>1</sup> And, therefore, it may suffice to confine attention to one or two important questions upon which the results throw light, and also to examine what at first sight is an insuperable difficulty. The former are the reproduction of the Metaphyta, the facts of mimicry and protective coloration, and the principle of "*panmixie*," and the latter is what is known as the Mendelian principle, after Gregor Mendel, whose experiments in intercrossing peas, in Bateson's words, "are worthy to rank with those that laid the foundation of the Atomic laws of Chemistry."

To the embryologist two of the most striking features about the higher plants are their powers of variability and the enormous gulf in organisation between the highly differentiated sporophyte and the lowly, insignificant gametophyte or sexual generation. As already written by the writer, "the plan carried out in animals has been such as to favour and foster the ever greater and greater amplification of the sexual generation. In plants, as elsewhere already insisted, the reverse is the case. Here the asexual generation has undergone increased amplification without ever being able to attain any very high degree of histological differentiation. The sexual generation of plants is at the best a miserable failure from the morphological point of view. . . . The higher one ascends the smaller it becomes, until, in the highest flowering plants it has almost reached the vanishing-point, without, however, being able to disappear entirely." Whilst throughout the Metazoa the duplicated number of chromosomes is retained everywhere except in the gametes and in their immediate forerunners, the oocytes and spermatocytes, in the Metaphyta the reduction of the chromosomes, and with this the elimination of characters, is carried out prior to the appearance of the sexual generation. The cells of this contain, therefore, the reduced number of chromosomes and characters, and possibly for this reason they possess no powers of variation.

But in the sporophyte, the duplication of characters is retained, and this exhibits, not only an immense capacity of adaptation, but also enormous powers of variation. A particular variety can be continued by cuttings, because its cells contain exactly the same duplicated qualities which gave the stamp to the variety itself at the start. And what light, on

<sup>1</sup> For this reason no reference is made to, for example, the "transmutation-theory" of de Vries.

the other hand, do not the presence of this duplication and its import throw upon the phenomena described under bud-variation by Darwin in chapter xi. of "Animals and Plants under Domestication"? In the words of Noll, previously cited, "Out of the embryonic substance of that lime-tree of Neustadt every year new leaves and buds form, but these remain in connection with the dying remains of structures of earlier years." Practically, the original apical cell (or cells), which formed each bud, is a continuation of the two sets of qualities present in the fertilised egg, but here in the bud they have been joined together through a varying but great number of cell-generations, and during this time they have been under the influence of the environment.

As Darwin writes: "The difference between seminal and bud-reproduction is not so great as it first appears; for each bud is, in one sense, a new and distinct individual; but such individuals are produced through the formation of various kinds of buds without the aid of any special apparatus, whilst fertile seeds are produced by the concurrence of the two sexual elements. The modifications which arise through bud-variation can generally be propagated to any extent by grafting, budding, etc., and occasionally even by seed. Some few of our most beautiful and useful productions have arisen by bud-variation" (21). And, again, on the next page: "These cases (of bud-variation) prove that those authors who, like Pallas, attribute all variability to the crossing either of distinct races, or of individuals belonging to the same race but somewhat different from each other, are in error, as are those authors who attribute all variability to the mere act of sexual union. Nor can we account in all cases for the appearance through bud-variation of new characters by the principle of reversion to long-lost characters. He who wishes to judge how far the conditions of life directly cause each particular variation ought to reflect well on the cases immediately to be given."<sup>1</sup>

In Weismann's recently published lectures nearly 70 pages are devoted to mimicry and protective coloration. Not unnaturally, it also plays a considerable rôle in the later consideration of his theory of "germinal selection." Looked at in the light of the views here advanced, the subject is important in

<sup>1</sup> The discussion of bud-variation and its cause, given by Weismann on p. 439 *et seq.* of the "Germplasm," is too long to be cited here.

demonstrating *ad oculos* the all-powerful influence of the environment. It may be maintained that protective coloration, or mimicry, is not adopted in order to conceal an animal, or to imitate one, but that they are simply exaggerated instances of the attempts unconsciously made by living things to bring about complete harmony between themselves and the environment. Were our mental vision keener, we should recognise this throughout organic nature: here we perceive it, because the organisms concerned reflect to us, as it were from a mirror, a part of their environment. It is hardly conceivable how natural selection, or Weismann's complicated germinal selection with its chance combinations of ids or groups of characters, should bring about the observed results.<sup>1</sup> On the other hand, a sufficient as well as a simple cause of the phenomena may be found in the self-regulating mechanism described in this writing. This may suffice, not only for the explanation of mimicry and protective coloration, but also of those phenomena classed by Weismann under *panmixie*. It is clear why the eyes of cave-animals, for instance, should tend to degenerate, for here the environment is one of constant absence of light. This being so, there is no stimulus present to keep up those characters or qualities necessary for the existence of organs of vision, and, without the assumption of the absence of a "life or death" natural selection, or the presence of *panmixie*, they degenerate, simply because, when Nature places narrow bounds upon the environment of an organism, she limits its organs.<sup>2</sup> Another illustration of the supposed action of *panmixie* is frequently given and discussed by Weismann. It is the disappearance of the hind limbs of the Greenland whale with the exception of the ischiatic bones.

<sup>1</sup> The known facts concerning mimicry depend to a large extent upon the coloration of the wings of insects. Quite recently (*CR. Acad.*, Paris, vol. cxxxvii. p. 1128, 1908) Countess Maria von Linden has published further studies upon the coloration of the wings of butterflies. Chemically, the fundamental colour would appear to be red, and this is shown to be a product of chlorophyll, the green colouring matter of plants. The hæmoglobin of animals is also concluded to be a product of chlorophyll—a brilliant idea! All the other colours of the insect-wing are derived from the red colouring matter by oxidation or reduction, and the authoress remarks "the colour of the wings of the butterfly is a question of oxidation, and not a question of selection, as the Darwinians will."

<sup>2</sup> "Durch den Verlust eines Sinnesorganes verliert der Mensch einen Theil seiner Aussenwelt. Die Natur wird auch die Sinnesorgane beschränken, wenn sie die Aussenwelt eines Thieres in enge Grenzen setzt."—JOH. MÜLLER, "Die vergleichende Anatomie der Myxinoiden," pt. ii. p. 34.



Without *panmixie* the self-regulating mechanism will be a sufficient cause of the loss of these, while the ischia persist, because they serve for the attachment of the muscles of the penis or clitoris.

Lastly, there remain for brief consideration the phenomena made out by Mendel, and since confirmed by de Vries, Correns, Tschermak, Bateson and Saunders, and others, in his experiments in crossing peas. For some time these extremely interesting facts appeared to the writer to form an impassable hindrance in the way of his conclusions. No solution of the difficulty appeared possible; but, finally, the words of Mendel himself gave the clue to what I believe to be the meaning of his results. This clue has, indeed, been mentioned more than once in the present writing. By kind permission of Mr Bateson, whose name is now closely associated with Mendelian facts and factors, a short account of Mendel's work can be given in the translator's words. The experiments relate to the crossing of varieties of the pea, *Pisum sativum*. This is naturally a self-fertilising plant and its pollen is to all appearance only very rarely carried from flower to flower by insects. "These experiments of Mendel's were carried out on a large scale, his account of them is excellent and complete, and the principles which he was able to deduce from them will certainly play a conspicuous part in all future discussions of evolutionary problems. . . . For the purposes of his experiments Mendel selected seven pairs of characters as follows:—

"1. Shape of ripe seed, whether round, or angular and wrinkled.

"2. Colour of 'endosperm' (cotyledons), whether some shade of yellow, or a more or less intense green.

"3. Colour of seed-skin, whether various shades of grey or grey-brown, or white.

"4. Shape of seed-pod, whether simply inflated, or deeply constricted between the seeds.

"5. Colour of unripe pod, whether a shade of green, or bright yellow.

"6. Nature of inflorescence, whether the flowers are arranged along the axis of the plant, or are terminal and form a kind of umbel.

"7. Length of stem, whether about 6 or 7 ft. long, or about  $\frac{3}{4}$  to  $1\frac{1}{2}$  ft.

"Large numbers of crosses were made between peas differing in respect of *one* of each of these pairs of characters. It was found that in each case the offspring of the cross exhibited the character of one of the parents in almost undiminished intensity, and intermediates which could not be at once referred to one or other of the parental forms were not found.

"In the case of each pair of characters there is thus one which prevails to the exclusion of the other. This prevailing character Mendel calls the *dominant* character, the other being the *recessive* character. That the existence of such 'dominant' and 'recessive' characters is a frequent phenomenon in cross-breeding, is well known to all who have attended to these subjects.

"By letting the cross-breds fertilise themselves, Mendel next raised another generation. In this generation were individuals which showed the dominant character, but also individuals which presented the recessive character. Such a fact also was known in a good many instances. But Mendel discovered that in this generation the numerical proportion of dominants to recessives is on an average of cases approximately constant, being in fact *as three to one*. With very considerable regularity these numbers were approached in the case of each of his pairs of characters. There are thus in the first generation raised from cross-breds 75 per cent. dominants and 25 per cent. recessives.

"These plants were again self-fertilised, and the offspring of each plant separately sown. It next appeared that the offspring of the recessives *remained pure recessive*, and in subsequent generations never produced the dominant again. But when the seeds obtained by self-fertilising the dominants were examined and sown, it was found that the dominants were not all alike, but consisted of two classes: (1) those which give rise to pure dominants; and (2) others which give a mixed offspring, composed partly of recessives, partly of dominants. Here also it was found that the average numerical proportions were constant, those with pure dominant offspring being to those with mixed offspring as one to two. Hence it is seen that the 75 per cent. dominants are not really of similar constitution, but consist of 25 which are pure dominants and 50 which are really cross-breds, though like the cross-breds raised by crossing the two original varieties, they only exhibit the dominant character.

"To resume, then, it was found that by self-fertilising the

original cross-breds the same proportion was always approached, namely: 25 dominants, 50 cross-breds, 25 recessives, or 1D : 2DR : 1R.

"Like the pure recessives, the pure dominants are thenceforth pure, and only give rise to dominants in all succeeding generations studied.

"On the contrary the 50 cross-breds, as stated above, have mixed offspring. But these offspring, again, in their numerical proportions follow the same law, namely, that there are three dominants to one recessive. The recessives are pure like those of the last generation, but the dominants can, by further self-fertilisation, and examination or cultivation of the seeds produced, be again shown to be made up of pure dominants and cross-breds in the same proportion of one dominant to two cross-breds.

"The process of breaking up into the parent forms is thus continued in each successive generation, the same numerical law being followed so far as has yet been observed. Mendel made further experiments with *Pisum sativum*, crossing pairs of varieties which differed from each other in *two* characters, and the results, though necessarily much more complex, showed that the law exhibited in the simpler case of pairs differing in respect of one character operated here also.

"In the case of the union of varieties, AB and ab, differing in two distinct pairs of characters, A and a, B and b, of which A and B are dominant, a and b recessive, Mendel found that in the first cross-bred generation there was only one class of offspring, really AaBb. But by reason of the dominance of one character of each pair these first crosses were hardly if at all distinguishable from AB. By letting these AaBb's fertilise themselves, only *four* classes of offspring seemed to be produced, namely :—

AB	showing both dominant characters
Ab	„ dominant A and recessive b.
aB	„ recessive a and dominant B.
ab	„ both recessive characters a and b.

"The numerical proportions in which these classes appeared were also regular, and approached the ratio :—

9AB : 3Ab : 3aB : 1ab.

"But on cultivating these plants and allowing them to fertilise themselves it was found that the members of the

## RATIOS

- |     |   |
|-----|---|
| 1   | ab class produce only ab's.             |
| 3 { | 1 aB class may produce either all aB's, |
| 2   | or both aB's and ab's.                  |
| 3 { | 1 Ab class may produce either all Ab's, |
| 2   | or both Ab's and ab's.                  |
| 9 { | 1 AB class may produce either all AB's, |
| 2   | or both AB's and Ab's,                  |
| 3   | or both AB's and aB's,                  |
| 4   | or all four possible classes again,     |
|     | namely, AB's, Ab's, aB's, and ab's,     |

and the average number of members of each class will approach the ratio 1 : 3 : 3 : 9 as indicated above.

"The details of these experiments and of others like them made with *three* pairs of differentiating characters are all set out in Mendel's memoir."<sup>1</sup>

For the information of the non-zoological reader it may be added, that Mendelian results have been obtained in some cases in animals, for instance, by Guaita, and by Bateson and Saunders (*vide* Report I. to the Evolution Committee of the Royal Society, London, 1902).

Turning now to an examination of the bearings of the Mendelian facts and finds upon the question of variation, it is not the latency of certain characters, or the dominance of others in Mendelian cases, like those of the peas, which to my mind raises any difficulty; it is the *non-elimination and disappearance of characters* which requires explanation, and their constant and regular reappearance in the offspring of the self-fertilising crosses, and, moreover, in proportions which follow the mathematical laws of probability. Obviously, if the writer's conclusions were correct, the Mendelian cases must be capable of elucidation. And on the other hand, if these were insoluble, the views here advanced could not be maintained. For long the difficulty

<sup>1</sup> Bateson, W. "Mendel's Principles of Heredity," Cambridge, 1902, *loc. cit.* p. 8-12. The full memoir should be studied. A translation of the original paper will also be found in *Journ. Roy. Hortic. Soc.*, 1901, and the original paper in German has been reprinted in Ostwald's "Klassiker, etc."

appeared to be insuperable; and yet, taking but simple cases, like the green and yellow cotyledons, the starting-point had been the union of only two characters, and in all subsequent generations after the first these were repeated according to the proportion of 1D : 2DR : 1R in the subsequent generations. The one character, therefore, was as good as the other, except that the one character was dominant or "prepotent." "Why," it was asked, "is there no elimination here?" Finally, the answer came in Mendel's own words. "No one will seriously maintain that the development of plants in the open country is ruled by other laws than in the garden-bed. *Here, as there, changes of type must take place, if the conditions of life be altered, and the species possess the capacity of adapting itself to its new environment.*" And again, a little further on: "Even here (*i.e.* in certain Leguminosae like and including *Pisum*) there have arisen numerous varieties during a cultural period of more than 1000 years; *these maintain, however, under unchanging environments, a stability as great as that of species growing wild.*"<sup>1</sup>

As the environment was constant, each of the characters dealt with was equally suitable and adapted to it, and no one could be selected to the exclusion of the other. Therefore, each would be equally favoured, and the results would come about according to the mathematical laws of probability, which Mendel and his successors found to be the case. The reappearance of characters in this way, in man and other living things, will often be brought to pass by the like cause, the absolute equality of the characters or qualities under an environment which has been constant, or which, at all events, has not caused any of them to change. It is this, with dominance or prepotency (a kind of close-linking together of characters in the pack), which accounts for the phenomena of "atavism," or of "reversion" to, for instance, a grandparent.<sup>2</sup> When, as already written in earlier pages, the

<sup>1</sup> Bateson, W. "Mendel's Principles," p. 82, 88. Not italicised in original.

<sup>2</sup> The phenomena and conclusions briefly mentioned in the above are of the kinds which Weldon and others refuse to consider except by an "appeal to ancestry." The writer ventures to think that embryology furnishes better evidence regarding the value of ancestry than does mathematics, and certainly the facts of embryology do not support the view that much, or anything, is to be gained from an "appeal to ancestry." In the union of egg and sperm we witness the joining-together of *but two sets* of characters, and not that of "x" sets, derived from as many "ancestors." In the development of the individual only *one* of these becomes manifest, but the other may re-appear in the gametes. As in the reproduction of

reduction (election and elimination) is merely the undoing of the previous union, it must be a matter of the mathematical probability which of the two halves should be chosen, for one must be eliminated; and thus, on occasion, the sperm or the egg may mimic, even in minute details, the characters or qualities of some near "ancestor," for instance, of a grandparent.

Possibly there are difficulties which, under the views advanced in the present writing, cannot be explained. If so, in spite of much search in the works of Darwin, Wallace, Weismann, Bateson, and Ewart, they are at present unknown to the writer. So far as he can see, the interaction<sup>1</sup> between the duplicated sets of characters and the environment, forming the self-regulating mechanism, offers a simple construction of all the phenomena of variation, an ultimate and a far more natural one than natural selection, or the germinal selection of Weismann. Indeed, under it there is no necessity to invoke these: *by it their positions are completely and decisively outflanked, and rendered untenable!*

In studying the mode of development, a thing which has naturally required a wide basis of observation and inquiry, nothing has struck the writer more than the obvious presence of rhythmic changes everywhere. From whatever aspect it be regarded, Life ebbs and flows. And is it at all likely that the rhythm of cell-division, of all reproductive phenomena, should

---

dioecious individuals these unite with the gametes of other individuals, in this union, *a priori* there would be for any particular character four possibilities. This may be illustrated. Take the four grandparents, and consider only one character in the gamete of each. Assume, further, that in the two following generations there be no complete elimination of any character and that this reappear in some of the gametes. The characters may be called A, b, C, d, the first two being conjoined in the germ-cells of the father, the second two in those of the mother, and the large letters being dominant or prepotent. In the gametes of the parents the representative of this character may be either the dominant one or the latent one. That is to say, any particular gamete may contain any one of the four characters, A, b, C, d, and suppose then that b and C be conjoined to form the grandchild, and that C be dominant in it, its gametes will then contain either character b or C. And therefore, if this gamete b unite with another e, and if in the development b be the dominant one, the result will give the appearance of a "reversion" to the grandparent b, after a dormancy through two generations. But there is no room for more "ancestors."

<sup>1</sup> Compensation and balance in the individual, man for example, may depend on *dual brain-action*, working as two individualities, and reacting to and endeavouring to compensate external and internal phenomena. For interesting evidence and quite similar conclusions, see Lewis C. Bruce (in *Brain*, 1895, and *Scot. Med. and Surg. Journ.*, 1897), "Dual Brain Action, etc."

have nothing comparable to it in the history of varieties or species? In the self-regulating mechanism of the present writing a physical cause of variation, etc., has been found, and one may look for something to bring it into vigorous action at fairly regular, even rhythmic, intervals. Notwithstanding the imperfection of the geological record, there is in it undoubted evidence of progress in an upward direction in the world of organisms. There has been, perhaps, a more or less rhythmic deposition of the rocks, and a regular appearance of new and higher forms. If the reader should consult the "Diagram representing the variations in the eccentricity of the earth's orbit for three million of years before 1800 A.D. and one million of years after it," on p. 312 of Croll's "Climate and Time in their Geological Relations," an actual physical basis for regular changes in climate at long intervals may be recognisable. As Croll has demonstrated, the rhythmic variations in the eccentricity of the earth's orbit, while not *directly* inducing change of climate, *indirectly* bring this about, and at regular intervals. With every long beat of this rhythm the self-regulating mechanism must be called into vigorous action if a variety or species shall survive under the new conditions, and under such new species must come into existence. Furthermore, whenever new species appear with change of environment, in course of time new varieties will arise from them. And whenever a new variety has appeared, as long as it is, or possesses characters, adapted to the environment, it and these will persist, for there can be no "swamping effects of intercrossing" with characters suited to a particular environment, as it is inconceivable that any of these should be eliminated in the germ so long as they are useful.

Not only is the self-regulating mechanism<sup>1</sup> a cause, nay, *the cause* of variation, and of the occurrence of varieties, which, as Wallace observes, arise to fill in all the available places, but it becomes a potent factor in the origin of species. If, for example, there be five varieties of a species, A, B, C, D and E, and if A and E be supposed to be the extreme ones, if there be a change in the environment, such that it become unsuitable for

<sup>1</sup> It may be noted as revealing the true inwardness of the writer's little track of research during more than fifteen years, that his work has resulted in the laying bare of *three self-regulating mechanisms*, to wit, that governing the span of gestation and the cause of birth, that regulating the proportion of the two sexes, and, finally, that underlying the cause of variation.

the three varieties B, C and D, and if these be unable to vary sufficiently to meet this change and compensate it, while A and E can do so, B, C and D will be eliminated, even if they be fertile with the two favourable varieties A and E. The two latter will be left, and having still further varied, they would now, because discontinuous, be regarded as distinct species. It is only when a variety or varieties become utterly incapable of adaptation to the environment that new species can arise. For, in the words of a great zoologist, August Weismann, "Alles an den Lebewesen beruht auf Anpassung."

#### XI. THE ENVIRONMENTAL MOMENT.

Of recent years, by many zoologists at all events, far less import has been attributed to the influence of the environment than to other and inner factors. Weismann's doctrines of *panmixie* and germinal selection might almost be said to dispense with the environment as a factor. Indeed, does not Weismann emphatically say of his germinal selection that "quite independently of the relations of the organism to the external world it creates variations"?<sup>1</sup> And as he, like other zoologists, has not recognised the existence of any germinal elimination, such as described in previous chapters, the real import of the environment under his views is that of a weeding-out of certain individuals and the selection of others owing to the struggle for existence and the survival of the fittest. The founders of the doctrine of natural selection, Darwin and Wallace, assigned little import to the environment.<sup>2</sup> For Buffon and Lamarck, and in recent years for Semper, Thiselton-Dyer,<sup>3</sup> and Ewart, it was a factor of immense gravity. And, of course, for the followers of Lamarck,

<sup>1</sup> Weismann, A. "Vorträge über Descendenz-Theorie," vol. ii. p. 134: "Darin gerade liegt die hohe Bedeutung dieses Kräftespiels im Keimplasma, dass es ganz unabhängig von den Beziehungen des Organismus zur Aussenwelt Variationen schafft."

<sup>2</sup> Wallace disputes any direct influence of the environment in causing variation (compare "Darwinism," pp. 418, 426, 436). On the other hand, Darwin wrote to Dr Moritz Wagner (Oct. 18, 1876): "In my opinion the greatest error which I have committed has been not allowing sufficient weight to the direct action of the environment, i.e. food, climate, etc., independently of natural selection."

<sup>3</sup> Thiselton-Dyer, W. T. "Variation and Specific Stability," in *Nature*, vol. li, Mar. 14, 1895, p. 459-461. Concerning the rôle of the environment, Thiselton-Dyer's conclusions closely resemble those drawn by the writer. He writes (p. 459): "I infer, therefore, and all the facts which have come under my observation confirm it, that a change in the external conditions, otherwise the *environment*, will provoke



represented by Cope, Osborn, Perrier, Ryder and others, who have upheld, and still maintain, the inheritance of acquired characters, the environment has been the only factor in producing variation.

Under the views here advanced it is, perhaps, possible to reconcile the two extremes, or at all events to indicate the outlines of a compromise. The Darwinians, with Weismann at their head, have been quite in the right in denying an inheritance of acquired characters. Clearly this is so, because, as a matter of fact, there is no inheritance or direct handing-over of anything from the individuals to the germ-cells, which form the next and all succeeding generations. On the other hand, the facts observed often give the appearance of an inheritance of acquired characters without this being at all real. Undoubtedly, the environment is all-powerful for the individual. Directly, its effects on the individual are not handed on, but these influences are reflected on its germ-cells. Everything in the life of the individual is also of consequence for the weal and woe of its germ-cells. The total environment, represented by climate, nutrition, disease, toxins and other factors, leaves its impress on the individual, *and on its germ-cells too*. Therefore, though there be no handing-on of acquired characters, the result, as already stated on an earlier page regarding the line of heredity, may be practically the same as though there had been.

The giraffe, for example, has a long neck, not because its ancestors were in the habit of stretching their necks, as the Lamarckians maintain, but because in this case from generation to generation Nature eliminated, not by natural selection those individuals whose necks were short, as the Darwinians assert, but those characters or qualities in the germ-cells which tended to the production of a shorter neck than did those characters or qualities of the other parental line, and she nourished and increased the importance of the latter from generation to

---

*some variation in the organism, which I may call the stimulated variation."* And again (p. 461): "When the environment varies, stability is destroyed, but it will be ultimately re-established, though with a different centre, by the operation of natural selection. The result is that the organism has undergone some permanent degree of change. As I conceive the process, it is one of continuous adjustment of 'slight' variations on one side or the other. But it is important to keep in view that variation in the environment stimulates the variation in the organism which supplies the ultimate material for adjustment."

generation. There is no chance variation here, such as Weismann's germinal selection assumes. Sometimes chance does come in, as in the instance of the Mendelian peas, but this is because here it is a matter of indifference which of two characters should be eliminated, as both are equally adapted to the environment. It is not denied that a struggle for existence does take place in nature, but it is not so clear that it should always result in a survival of the fittest, for, as Wolff and others have insisted, the element of chance enters into it. In any case the result is not one which can induce genetic variation, or produce varieties, or give origin to new species. By the self-regulating mechanism, described in previous pages, Nature must in all sexual reproduction eliminate half of the groups of characters, half of the individualities. In its magnitude this is appalling, and it results in an election beside which natural selection is as nothing.

Of the individual, Galton long ago said that it was the trustee of the germ-cells. In the light of present results how true this is! In our social life the parent is made answerable more or less, for the well-being and education of his children. *Of this responsibility nothing whatever is assigned for the little insignificant, but for good and ill immensely potent, entities, the germ-cells!* While for the state, for the commonwealth, it may be—I do not say it is—a matter of indifference under what environment the individual pass his span of life; for the well-being of future generations, for the good of the race, it can only be of the utmost moment, that, as the germ-cells are the seed of the stock, contained in individuals, the total environment should be made, so far as it is possible, of the healthiest and best description for the latter. The weekly or yearly table of death-rate is no real index of national improvement or deterioration, for even degeneration and longevity may go hand in hand.

In this connection the recent report of the Royal Commission on Physical Training (Scotland)<sup>1</sup> furnishes matter for serious reflection.

<sup>1</sup> 1903, "Cd. 1507." Here various comparisons are made between the children of Edinburgh and Aberdeen, and their respective conditions of existence, etc. In these cities "the balance, in respect of health and development," was found to be "in favour of children drawn from three and four (and upwards) roomed houses"; and Aberdeen, "most of whose children were drawn from three-roomed houses, had the advantage over Edinburgh, whose children were drawn mostly from two-

In considering the welfare of the race, it would not be wise to lay stress on the fact that, where necessary, Nature eliminates the unsuitable; for to permit of this, it must be possible to prevent the mating of the unfit with the unfit. Rather, let it be borne in mind that, in the words of H. G. Wells, "Nature is a reckless coupler . . . and she slays," and that on occasion she may remorselessly destroy, not merely the total individuals of a variety, but even of a species.

"'So careful of the type?' but no,  
From scarped cliff and quarried stone  
She cries, 'A thousand types are gone:  
I care for nothing, all shall go.'"—TENNYSON.

Our highly-civilised life in large towns and cities is one in which "the conditions of natural existence" (Semper) too often become the reverse, for the wealthy scarcely less than for the poor. At both ends of the scale reform is urgently called for. The higher classes of society are not recruited to any great extent from among themselves, but from those, the middle classes, beneath them. A most significant fact! The population of cities is recruited from the country, just as the middle classes reinforce the aristocracy. The rural and middle classes are, and must be, the mainstay of a nation; for with them there are the best attempts at adaptation to the environment. But "the poor we have always with us," and the great problem for the reformer, for the city rulers, aye, for the statesman, is how to make their total environment such that, instead of deteriorating, in them the stock shall improve.

To the worker the practical application of his results is denied: with the solution of the problem his task is done. Statesmen have various ambitions, and at times to every civilised

---

roomed houses." "Mental dulness was noted in 8·8 per cent. of the Aberdeen children, compared with 12·33 per cent. in Edinburgh." On p. 25 the Commissioners say: "No great amount of argument is required to bring home to everyone the significance of such facts as these. Height, weight, pallor, bad health, bad nutrition, want of alertness, and bad carriage, besides other conditions not noticed here but dealt with in the reports, distinguish Edinburgh adversely as compared with Aberdeen; and even Aberdeen, which probably is fairly representative of Scotland generally, as compared with the standard of the British Isles, leaves something to be desired." Indeed, the whole report reflects the all-powerful influence of the conditions of existence to him who can read between its lines, in as clear a fashion as do the facts of mimicry.

nation the man is given whose single aim is the welfare of his people. Fortunate the race that breeds such! For beyond measure is the greatness of him who shall achieve success in this task!

## REFERENCES.

16. Rauber, A. "Der Ueberschuss an Knabengeburten und seine biologische Bedeutung," Leipzig, 1900, *loc. cit.* p. 142.
17. Lenhossék, M. von. "Das Problem der geschlechtsbestimmenden Ursachen," Jena, 1903, *loc. cit.* p. 81.
18. Ewart, J. C. "Variation; Germinal and Environmental," in *Trans. Roy. Dublin Soc.*, vol. vii. p. 353-378, 1901.
19. Bateson, W. "Mendel's Principles of Heredity," 1902, p. 200.
20. Delage, Yves. "L'Herédité," 2nd ed., 1903, *loc. cit.* p. 778.
21. Darwin, C. "Animals and Plants under Domestication," vol. I. p. 373, 1868.

NOTE.—With reference to p. 133, see an important paper by Max Hartmann (Die Fortpflanzungsweisen der Organismen) in *Biol. Ctrbl.*, vol. xxiv., 1904.

## LIST OF MEMOIRS UPON THE METAZOAN LIFE-CYCLE, 1889-1903.

1. The Early Development of *Lepidosteus osseus*, in *Proc. Roy. Soc.*, London, Vol. 46, p. 108-118, 1889.
2. The Transient Ganglion-Cells and their Nerves, in *Anat. Anz.*, Vol. 7, p. 191-206, 1892.
3. On a Supposed Law of Metazoan Development, in *ibid.*, Vol. 8, p. 22-29, 1892.
4. On the Phenomena of Reproduction in Animals and Plants, on Antithetic Alternation of Generations, etc. (with J. A. Murray), in *Ann. Botany*, Vol. 9, p. 441-468, 1895; also in *Anat. Anz.*, Vol. 11, p. 234-255, 1895.
5. The History of a Transient Nervous Apparatus in certain Ichthyopsida: An Account of the Development and Degeneration of Ganglion-Cells and Nerve-Fibres, Pt. 1, *Raja batis*, in *Zool. Jahrb. Morph. Abteil.*, Vol. 8, p. 1-106, 8 plates, 1896.
6. Further Remarks upon the Phenomena of Reproduction in Animals and Plants, in *Anat. Anz.*, Vol. 11, p. 634-641, 1896.
7. On Certain Problems of Vertebrate Embryology (The Critical Period, etc.), p. 1-77, Jena, Gustav Fischer, 1896.
8. The Yolk-Sac, Yolk, and Merocytes in *Scyllium* and *Lepidosteus*, in *Anat. Anz.*, Vol. 14, p. 334-347, 1896.
9. On the Disappearance of the Transient Nervous Apparatus in the Series: *Scyllium*, *Acanthias*, *Mustelus*, and *Torpedo*, in *ibid.*, Vol. 12, p. 371-374, 1896.
10. The Span of Gestation and the Cause of Birth, p. vii. and 132, Jena, Gustav Fischer, 1897. (Abstract of the foregoing in *Anat. Anz.*, Vol. 14, p. 97-102, 1897, under the title: "The Rhythm of Reproduction in Mammalia.")
11. The Birth-Period of *Trichosurus vulpecula*, in *Zool. Jahrb. Morph. Abteil.*, Vol. 11, p. 77-96, 1897, 1 plate.

12. The Morphological Continuity of the Germ-Cells in *Raja batis*, in *Anat. Anz.*, Vol. 18, p. 485-485, 1900.
13. The Germ-Cells of *Pristiurus*, in *ibid.*, Vol. 21, p. 50-61, 1902.
14. The Numerical Law of the Germ-Cells, in *ibid.*, Vol. 21, p. 189-200, 1902.
15. The Germ-Cells, Pt. I., *Raja batis*, in *Zool. Jahrb. Morph. Abteil.*, Vol. 16, p. 615-702, 2 plates, 1902. Also in course of republication in *Journ. Anat. & Physiol.*, Vol. 38, 1903-04.
16. Heredity and the Epicycle of the Germ-Cells in *Biol. Ctrbl.*, Vol. 22, p. 321-328, 353-360, 398-408. Also in *Trans. Bot. Soc.*, Edinburgh, 1902.
17. The Determination of Sex in Animal Development, in *Zool. Jahrb. Morph. Abteil.*, Vol. 16, p. 703-764, 1 plate and 3 text figures, 1902. Like Nos. 7 and 10 this has been separately published by Herrn Gustav Fischer, (Jena). Abstract of the above in *Anat. Anz.*, Vol. 20, p. 556-561, 1902.
18. Embryological Aspects and Etiology of Carcinoma, in *Lancet*, June 15, 1902, p. 1758-1761.
19. The Embryology of Tumours, in *Anat. Anz.*, Vol. 23, p. 486-494, 1903) Also in German under the title, "Die Embryologie der Geschwülste," in *Ctrbl. all. Path. u. Path. Anat.*, Vol. 14, p. 513-520, 1903. (See also "Ueber Embryomata und andere Geschwülste," in *Berliner klin. Wochenschr.*, 1903, No. 30, and for full paper, *Journ. Anat. & Physiol.*, 1904.)
20. The Germ-Cells, Pt. II., *Pristiurus melanostomus*, with 1 plate and 14 figures in text. (In the Press.)

---

## Abstracts

### ANATOMY.

**THE PARIETO-OCCIPITAL FISSURE.** J. FROUDE FLASHMAN,  
(66) *Reports from the Pathological Laboratory of the Lunacy Department*  
(N.S.W.), Vol. i., Part 1, 1903.

UNTIL three months ago there was practically no literature relating to the morphology of the parieto-occipital fissure. Since then no less than four independent memoirs dealing with this subject have been published. In the most recent part of Schwalbe's *Zeitschrift für Morphologie und Anthropologie* (Bd. iv., Heft 2, 1903), Kohlbrugge and Zuckerkandl both discuss the morphology of the parieto-occipital fossa in the apes: in the current number of the *Journal of Anatomy and Physiology* (January 1904), I have summarised the results of a comparison of the constitution of this fossa in man and the apes: and in the note under review, Flashman has studied it from a comparative human standpoint.

Flashman's ingenious interpretation of the morphology of the

cortical area surrounding the parieto-occipital fossa can, I believe, only be applied to a very limited number of cases. The chief interest of his work is the fact that, without any special knowledge of the Simian brain, he has selected as the primitive form of parieto-occipital fossa in the brain of the aboriginal Australian the same type as I had independently chosen after comparing the brains of a series of Chimpanzees and Gorillas with those of a number of exceptionally pithecoïd African brains. This undoubtedly primitive type presents, in place of one depression or "fissura parieto-occipitalis," three independent sulci, separated by an arcuate gyrus. Two of these sulci form a vertical Y-shaped pattern on the mesial surface of the hemisphere; whereas the third notches the dorso-mesial edge between the two limbs of the Y and often joins the intra-parietal sulcus on the dorsal surface.

Both Flashman and I have independently come to the conclusion that it is the anterior limb of the Y—my "sulcus limitans præcunei"—which becomes the chief element in the fossa parieto-occipitalis of most human brains. Flashman believes that in the development of the higher from the lower type of human brain the area containing the intermediate element—*incisura parieto-occipitalis mihi*—and the caudal end of the intra-parietal sulcus becomes rotated from the dorsal on to the mesial surface of the hemisphere behind the sulcus limitans præcunei (*mihi*), and that the latter becomes the parieto-occipital fissure of most writers.

I have attempted to show that the three independent sulci are usually (at any rate in Egyptian brains) buried in the one great depression—*fossa parieto-occipitalis*. G. ELLIOT SMITH.

**THE FOSSA PARIETO-OCIPITALIS.** G. ELLIOT SMITH, *Journ.* (67) *Anat. and Physiol.*, Vol. xviii., p. 164.

WITHIN the internal (mesial) parieto-occipital fissure, the author describes a concealed gyrus forming a bridge between the margins of the fissure, to which he gives the name of "*Arcus intercuneatus*," and which he considers to represent the primitive condition of the human brain. He also traces its development among apes up to the human form, but only as a preliminary to a wider study.

DAVID WATERSTON.

**DESCRIPTION OF SULCI OF FOUR BRAINS OF AUSTRALIAN**  
(68) **ABORIGINES.** J. F. FLASHMAN, *Reports from the Pathological Laboratory of the Lunacy Department (N.S.W.)*, Vol. i., Part 1, 1903.

So far as I am aware, all that is known of the brain of the aboriginal Australian is comprised in the descriptions of in-

dividual specimens by Rolleston (*Journal of the Anthropological Institute*, 1888) and Karplus (Obersteiner's "*Arbeiten*," 1902), and the note on the "Affenspalte" in six Australian brains by Duckworth in *Nature* (December 3rd, 1903). Flashman is thus the first writer to describe all the sulci in a series of brains.

G. ELLIOT SMITH.

**A NOTE ON AN EXCEPTIONAL HUMAN BRAIN PRESENT  
(69) ING A PITHECOID ABNORMALITY OF THE SYLVIAN  
REGION.** G. ELLIOT SMITH, *Journ. Anat. and Physiol.*, Vol.  
xviii., Pt. 2, p. 158.

THE specimen described is the left cerebral hemisphere of an Egyptian male fœtus about 33 weeks old, in which the anterior limiting sulcus of the island of Reil was prolonged upwards beyond the insular region, in the same way as the fronto-orbital sulcus in several genera of anthropoids, and was separated from the dorsal limiting sulcus by a narrow submerged gyrus.

The specimen showed marked abnormalities in other respects which are not described, and the author states that it is the sole example of the described variation which he has met with in over 1200 Egyptian hemispheres. The paper is an anticipation of the results of a forthcoming memoir on the Sylvian fissure.

DAVID WATERSTON.

**SOME METHODS FOR THE PERMANENT PRESERVATION OF  
(70) GOLGI PREPARATIONS UNDER A COVER-GLASS.** C.  
QUAIFE, *Reports from the Pathological Laboratory of the Lunacy  
Department* (N.S.W.), Vol. i., Part 1, 1903.

IN this interesting and valuable contribution, Mr Quaife, who is a student of medicine in the University of Sydney, describes four modifications of the Golgi method of colouring nerve tissue which permit the specimens to be mounted in Canada balsam under a cover-glass without suffering any deterioration.

The first method consists of staining the tissue according to Cox's directions; then embedding in celloidin (for which the author gives minute instructions), and cutting the sections; placing the sections successively in solutions of (1) hydroquinone, 2 parts; cryst. sodium carbonate, 5 parts; cryst. sodium sulphite, 5 parts in 100 parts of water; then (2) 10 per cent. sodium thio-sulphate; and (3) after washing, put in ammonium sulphide 5 per cent. in water. After washing well, the sections may be mounted under a cover-glass, because the deposit of insoluble sulphide of mercury is of such a stable nature that the specimen is permanent.

In the second method, specimens treated by Cox's or the ordinary sublimate procedure are placed successively in fluids 1 and 2 of

the first method; then they are placed in a solution of sodium thiosulphate, 20 parts; cryst. sodium sulphite, 5 parts; 5 per cent. gold chloride, 10 parts; and water up to 100 parts; and after twenty-four hours the sections are transferred to 10 per cent. hypo. solution for thirty minutes.

A deposit of metallic gold is the result of this procedure.

In the third modification, tissues stained by either the slow or the quick silver method are treated so as to convert the deposit into sulphide of silver; and in the fourth modification the deposit formed by the slow silver method is converted into metallic silver.

For the details the original must be consulted. Specimens which had been prepared according to these methods, and mounted under cover-glasses, were found not to have deteriorated after nine, or in some cases thirteen months.

G. ELLIOT SMITH.

**NEURONOPHAGIA.** G. ESPOSITO, *Il Manicomio*, No. 3, 1902, and (71) No. 2, 1903.

THE author's investigations were made chiefly upon tissues from a case of rabies in a child, but also upon other material from the human subject and from lower animals. They confirm certain of the conclusions of Cerletti (see *Review of Neurology and Psychiatry*, December 1903, p. 799).

Although the glia cells which normally lie in the vicinity of the nerve cell may, when the latter tissue element suffers morbid change, become more prominent and increase in number, there is no satisfactory evidence that they assume a distinct phagocytic action. The appearances presented by disintegrating nerve cells consist better with the view that they are simply undergoing a process of solution and absorption, which is probably determined by the action of special secretions of the glia cells and leucocytes. It is only very rarely that the leucocytes themselves emigrate into the nervous tissues. The process by which the removal of degenerated nerve cells is effected is to be regarded as one of neuronolysis rather than as one of neuronophagia.

W. FORD ROBERTSON.

## PHYSIOLOGY.

**EXPERIMENTAL PROOF OF THE EXISTENCE OF TROPHIC (72) NERVES.** G. PAGANO, *Riv. di patol. nerv. e ment.*, f. 1, 1904.

THE author remarks that although the question whether the nervous system has a direct influence on the nutrition of the tissues is one that has been much discussed, all the researches undertaken for the purpose of determining it have as yet been in vain. It is not the fact of a general action of the nervous system



on the nutrition of the tissues that is in question, but the means by which this action is effected, or the existence of special trophic nerves. The difficulty has been to obtain evidence of the occurrence of a purely trophic disturbance, in the production of which the action of motor, sensory or vaso-motor disorders could have played no part.

After numerous experiments, Pagano has succeeded in producing lesions which he holds to be purely of a trophic nature. The method employed was the injection of .25 to 1 c.c. of a 1 per cent. solution of prussic acid into the lumbar arachnoid of the dog. A complete motor, sensory and vaso-motor paralysis of the posterior part of the body is thereby rapidly produced. This passes off in about half an hour, the motor paralysis disappearing some minutes before the sensory paralysis. In a few hours the animal appears to have returned to the normal condition. About forty-eight hours later, however, there begin to appear, especially about the outer aspect of the thigh, a number of spots in which the hair rapidly sheds, or is easily pulled out, leaving an inflamed epidermis. These areas are often, on their first appearance, over an inch in diameter. They rapidly extend and deepen, so that within a few days destructive lesions of wide extent may be produced. Motor power and sensation remain unaffected. There is evidence that the lesions tend specially to occur on the side upon which the animal is accustomed to lie. External pressure seems, however, not to be necessary for their production. In cases in which the lesions are slight, repair commences in from eight to ten days, and generally goes on with moderate rapidity to complete reintegration of the cutaneous tissues. The deeper lesions are repaired more slowly and leave extensive cicatrices.

The author maintains that these experiments demonstrate the existence of special trophic nerves, upon which prussic acid is evidently capable of displaying a selective action. He mentions that the trophic lesions do not occur on the corresponding side if the abdominal sympathetic is cut, and he points out that this is in accord with the experimental evidence in regard to the production of corneal ulceration in consequence of lesions of the Gasserian ganglion and fifth nerve and its prevention by simultaneous section of the cervical sympathetic.

W. FORD ROBERTSON.

#### **A NOTE ON THE COMMUNICATION BETWEEN THE MUSCULO-**

(73) **SPIRAL AND ULNAR NERVES.** G. ELLIOT SMITH, *Journ. Anat. and Physiol.*, Vol. xviii., Pt. 2, p. 162.

A BRANCH was given off from the musculo-spiral high up, ran with the ulnar nerve to a point three inches above the elbow, and then entered the inner head of the triceps.

In the opposite limb, a portion of the ulnar nerve ran in the substance of the flexor carpi ulnaris muscle.

DAVID WATERSTON.

**ON THE SYMPATHETIC SYSTEM OF BIRDS, AND ON THE  
(74) MUSCLES WHICH MOVE THE FEATHERS.** J. N. LANGLEY,  
*Journ. of Physiol.*, Vol. xxx., 1903, p. 221.

THE investigations were carried out in pigeons, fowls and ducks. The anatomy of the sympathetic system is dealt with, and the results of stimulation of various parts recorded. From these the author concludes that the sympathetic system resembles that of mammals in the following particulars:—

The spinal sympathetic fibres arise from a localised region of the cord lying between and slightly overlapping the regions of origin of the nerves to the limbs. The efferent spinal sympathetic fibres in the cervical region, and probably in all others, end in connection with cells of the sympathetic ganglia. The post-ganglionic fibres run to the skin by way of the cutaneous branches of the spinal nerves. The areas of supply in successive cutaneous branches overlap very slightly. There are no commissural nerve cells in the ganglia, and there are probably no nerve cells giving off afferent fibres of the type of spinal ganglion cells. The pre-ganglionic fibres of successive spinal nerves supply successive but largely overlapping regions of the body.

The differences between the sympathetic systems of birds and mammals are:—

The spinal origin of pre-ganglionic fibres supplying the anogenital region in birds is not as in mammals in a portion of the cord which extends posteriorly of that giving origin to the fibres supplying the skin of the body. Spinal nerves send as a rule individual fibres to fewer ganglia than in mammals, most fibres are connected with the nerve cell of one ganglion only, hence the axon reflexes are more limited in the bird. The post-ganglionic fibres are medullated, and those of any one vertebral ganglion run more exclusively to the corresponding spinal nerve than they do in the mammal.

The feathers are depressed, raised and rotated by a complicated system of plain muscles supplied by sympathetic nerve fibres. The depressors are larger than the erectors, and stimulation of the mixed nerve supply usually causes depression, but the two muscles can be made to contract separately. Many of the areas of skin supplied by post-ganglionic and pre-ganglionic fibres of the spinal nerves are given.

Stimulation of the cervical sympathetic causes slight retraction of the eyelids, pallor of the mucous membrane of the mouth and

nose, with slight retraction of the mucous membrane of the latter, contraction of the cutaneous blood-vessels of the neck, and depression or occasionally erection of the feathers. It has no effect on the pupil.

Section of sympathetic fibres causes erection of feathers and dilatation of blood-vessels in the skin.

The muscles of the feathers are not paralysed by curara, and atropin, apocodeine, and adrenaline have little or no effect on them. Strychnine after curara causes irregular rhythmic depression and erection of the feathers by stimulating the spinal cord. Asphyxia usually causes prolonged depression of the feathers, but sometimes has a rhythmic effect like strychnine. Nicotine stimulates the sympathetic ganglia as in the mammal, but does not paralyse the pre-ganglionic fibres.

Lastly, the author gives a detailed description of the anatomy of the muscles of the feathers in various regions, and illustrates them by numerous diagrams.

PERCY T. HERRING.

#### STUDIES ON THE FORM OF THE ERGOGRAPHIC CURVE.

(75) LUIGI LUGIATO, *Riv. di Patolog. nerv. e ment.*, Vol. viii., Dec. 1903, p. 429.

THE author has made use of the ergograph to record the muscular curve in the same way as the myograph is ordinarily used for analogous researches in animals: this naturally led him to compare the two instruments, and to seek to make clear the advantages and disadvantages of the former. He refers to the criticisms on the ergograph made by Binet and Vaschide, and by Z. Treves, and to the new forms of ergograph constructed by these writers.

The paper is mainly concerned with the relation between the work done by raising the weight and the movement of the finger. Dr Lugiato has constructed a simple and ingenious instrument by means of which the exact position of the finger is indicated on the tracing. This consists of a rectangular piece of wood continued into the quadrant of a circle on which the angles of 0°, 22° 30', 45°, 67° 30', and 90° are marked by copper wire; an angle of 22° 30' in the negative direction being similarly marked on the rectangle. This instrument is fixed to the platform of the ergograph, so that the middle finger in the horizontal position is parallel to the line of 0°, the metacarpo-phalangeal joint corresponding to the right angle of the quadrant. A metal button is fixed to the leather loop which surrounds the finger; by this means an electric circuit is closed whenever the finger passes one of the marked angles, and by a special arrangement this is recorded on the tracing along with the ordinary curve. From an examina-

tion of the tracings thus obtained, two facts result. The first is that the weight does not begin to move simultaneously with the finger, but appreciably later. The second is that equal displacements of the weight do not correspond to equal displacements of the finger. Graphic demonstration of this fact is given by projecting the points marking the angles on a vertical line, and it is thus shown that the displacement of the weight is least during the first angle described by the finger, and becomes progressively greater. Professor Mosso had arrived at analogous results by means of experiments on the hand of a corpse. Thus it is seen that the work done by the finger in passing over equal spaces increases as the flexion increases, and in this respect, therefore, the ergograph is inferior to the common myograph, in which equal contractions of the muscle produce equal amounts of mechanical work.

If the tip of the finger be regarded as a point moving round the circumference of a circle, its motion may be resolved into a horizontal and a vertical displacement, and of these it is the horizontal displacement which mainly affects the movement of the weight. It is easily seen that this latter displacement (the abscissa) is very small for an arc of  $22^{\circ} 30'$ , and indeed when heavy weights are used, it may be counteracted altogether by a slight extension of the cord. This, in part, accounts for the observed fact that the weight does not begin to move simultaneously with the finger.

In some subjects the curve in the tracing is observed to descend when the finger begins to move. This can be placed in correlation with the fact that during the period of repose some individuals put their finger in a position of exaggerated extension, so that it approaches the angle of  $-22^{\circ} 30'$ . The weight is thus in a slightly raised position, and is lowered as the finger passes through the angle of  $0^{\circ}$ .

By the help of trigonometrical considerations Dr Lugiato arrives at the following formula to determine the amount of displacement of the weight in terms of the angle ( $\omega$ ) and radius of rotation ( $R$ ) of the middle finger and the length of the string ( $L$ ):—

$$\text{Displacement} = R + \frac{R^2 \sin^2 \omega}{2L} - R \cos \omega$$

The writer then proceeds to investigate the *force* expended by the finger in its motion. The magnitude of a force is proportional to the work done in a unit of *time*; therefore it was possible that the force might be constant, if to the increased work done as the flexion of the finger increased, had corresponded an increase in the time required. To facilitate this investigation a chronograph has been used in several of the experiments, and the points which

mark the passing of the finger through the angles of 22° 30', etc., have been projected on the chronographic tracing. It was found that the length of the periods of time thus marked off did not consistently correspond in any way to the amount of work done; sometimes a longer, sometimes a shorter amount of time being required at the beginning, when the amount of work done is least. In short, in this respect, every individual exhibits his own personal characteristics. This want of correspondence between the displacements of the weight and the time required to produce them, Dr Lugiato terms *variation of force*.

In spite of the imperfections he points out, the writer regards the ergograph as a marvellous instrument both for the simplicity of its construction and for the magnificent results it has already given and is yet destined to give in the investigation of work and of muscular contraction in man.

Among the many advantages of the ergograph, not least are the following: first, it does not alter the form of the curve, the pen, as well as the weight, moving in a straight, not in a curved line, as is usual in the myograph and the ordinary cylinder; second, the tracing it gives is not exaggerated by means of a lever, but corresponds perfectly to the displacement of the weight.

Moreover the ergograph adapts itself to many interesting investigations which it would be difficult to approach in any other way. Thus, for example, Dr Lugiato has made use of it in a study of the *time of latent contraction* and the time of *residual contraction*, the results of which he promises to give in another communication.

MARGARET DRUMMOND.

**M. BLONDLOT'S WORK ON THE N-RAYS, AND THE FIRST**  
(76) **RESEARCHES OF M. CHARPENTIER ON THE N-RAYS**  
**EMITTED BY THE ORGANISM.** H. GUILLEMINOT, *Arch.*  
*d'Electricité méd.*, Jan. 25, 1904, p. 51.

**RESEARCHES ON THE N-RAYS IN THE ORGANISM.**  
(77) AUGUSTIN CHARPENTIER, *Arch. d'Electricité méd.*, Jan. 25,  
1904, p. 58.

DURING the past year Blondlot has discovered and examined a new class of rays, which he has designated N-rays, from the initial letter of the town of his birth, Nancy. These rays emanate from a number of sources, in fact from any subject in a state of molecular tension; in particular may be mentioned muscle and nerve, especially in a state of activity. The rays, which are directly invisible, are best seen by their effect in increasing the fluorescence of a screen of platino-cyanide of barium, or of phosphorescent sulphide of calcium, the examination being conducted in a dark

room. When the screen is approached to a nerve centre, a nerve, or a muscle in action, its brightness increases. The rays behave for the most part in the same manner as rays of light as regards reflection, refraction, interference and polarisation. They can pass through aluminium, glass, and a solution of salt, but are arrested by lead, distilled water and damp paper.

In studying their action with the use of lead tubes of two to four inches in length, closed at one end by a thin slice of cork or cardboard rendered phosphorescent by sulphide of calcium, Professor Charpentier of Nancy found that as the open end of the tube (held next the body) is approached to a muscle in action, such as the heart, the phosphorescence increases when its area is reached. He found also that the course of a nerve can be traced out in a similar manner, the brightness of the light being increased when the nerve is approached.

Similar results are said to follow from examination of the various areas of the brain, the phosphorescence increasing as the body is placed over Broca's convolution of an individual who is speaking, or over the arm or leg areas when these limbs are put into a state of activity. Charpentier also finds that when the tube is placed over the forehead opposite the frontal lobes, the screen glows more brightly during the act of attention and mental effort. He claims that these remarkable results are not in any way due to the radiation of heat from the body, that being eliminated by the interposition of layers of non-conducting substance.

If these results are confirmed on further examination, their importance to physiology and medicine can hardly be over-estimated.

ALEXANDER BRUCE.

## PSYCHOLOGY.

**THE PHENOMENA OF PARAMNESIA.** *A propos of a special case.*  
(78) AUG. LEMAITRE, *Arch. de Psychol.*, Nov. 9, 1903, p. 101.

PARAMNESIA is the term applied to the illusion that something which has just been seen or felt for the first time has been experienced previously, but without, as a general rule, any accompanying idea of the *when*, the *how*, or the *where*. In the case studied by M. Lemaître, the subject knows the *where*—namely, at the same place; the *how*—namely, in a dream; and the *when*—at some date known approximately or even precisely.

The subject, who may be called Léon, is a young man of sixteen. He is myopic, and suffers from almost continuous headache. In childhood he was a somnambulist, and was subject to nightmares and hallucinations. At about the age of nine years he had a severe attack of scarlet fever, and his somnambulism

disappeared, but was replaced by, probably was transformed into, paramnesia, by a process not very difficult to explain.

To Léon himself the phenomena to be exemplified are without dubiety dreams which have become true. The following examples are in his own words:—

"I once dreamed that I was sitting on the slope at the side of a road, when a blackbird alighted just opposite me. I saw only the white surface of the road within a radius of 50 centimetres around the blackbird, with the end of a green branch above. I did not see myself, but I *felt* that I was in a sitting posture, with my arms hanging between my knees, my body inclined, and my hands closed. About a month after this I found myself in that very situation, and the moment the circumstances above narrated were accomplished, I remembered having dreamed it all before. I immediately noted the fact for you: 15th January, dream; 17th February, realisation.

"Another time I dreamed that I stepped on to the pavement before the milk shop of Vaudœuvres in the *Place des Philosophes*, and from the granite kerb where I was walking I looked into the shop of a tailor who has since removed. I dreamed this on the 12th of November; it came true on the 27th of December.

"During a French lesson (20th April) the black-board had at one time the exact appearance which it had presented in a dream about a month before—the same words, the same letters, even the chalk marks caused by the duster."

A number of examples of this kind are quoted. What explanation of them can be given? The explanation suggested by M. Lemaitre is that the experience which appears to Léon to have been passed through on some former occasion in a dream has, as a matter of fact, been passed through in reality, but without awaking his consciousness. In other words, paramnesia is the conscious revivification of a primary unconscious perception—of a perception which impressed the subconscious memory while the superior (conscious) centres were distracted or preoccupied. This theory will be best understood from the account given of an example of the phenomenon under consideration which occurred at Léon's first visit to M. Lemaitre. Léon suddenly, when in the middle of a sentence, got up from his arm-chair, turned round, and cried, as if awakening from a half-sleep, "You have just such a chair, the same draping, the same design, the same red flowers, as I saw in a dream a month ago. I have seen it all clearly before, but only from the seat to the arms." It is evident that there could be no question here of the throwing back (*antériorisation*) of perception without involving the absurdity of maintaining that Léon could see with his back. When his memory of flowers embroidered in a certain way on the drapery of the chair suddenly awakened,

and he interrupted himself to rise up, to turn round, and narrate what he thought he had dreamed, this was because his subconscious memory had spontaneously come into relationship with the higher psychic centres which had been occupied with something else, and the habitual phenomena of paramnesia asserted themselves. Léon took for an occurrence nearly a month old the memory of the chair-cover, subconsciously absorbed only a few moments before, when he was asked to seat himself in the chair.

It is to be remarked that in his "dreams" Léon sees and feels, but does not hear nor taste. Sounds and tastes may occur in the "realisation," but not in the premonitory "dream." When Léon was asked whether he ever noted the date of a dream before its realisation, he replied that he could not do so because he dreamed a great deal, and only a very few of his dreams came true.

W. B. DRUMMOND.

### **PATHOLOGY.**

#### **ON THE PATHOLOGY OF THE GLETS OF THE SENSORY**

(79) **GANGLIA.** E. LUGARO, *Riv. di Patol. nerv. e ment.*, Vol. v., f. 4, 6, and 9; Vol. vi., f. 10; Vol. vii., f. 3; Vol. viii., f. 11.

THIS work is a long and exhaustive research of 127 pages, and is so full of detail that a brief indication of the more important points is all that can be attempted in an abstract. The writer first gives the literature on the subject and then indicates his line of research, which is as follows: (1) Resection of the brachial plexus practised in rabbits and dogs. (2) Resection of the vagus in rabbits and guinea-pigs and cats. (3) Resection of the vagus and cervical sympathetic in rabbits, dogs and cats. (4) Resection of the vagus and extirpation of the superior cervical ganglion of the sympathetic in rabbits. (5) The last pair of spinal nerves were cut within the intervertebral foramina, a short distance from their respective ganglia in cats, rabbits and dogs.

Many fixatives were employed, and after trial the corrosive picric solution (equal parts of a saturated solution) was found the best. Toluidin blue proved to be the best stain, the subsequent differentiation taking place in absolute alcohol.

A counterstain such as erythrosin was always found to be objectionable. Delafield's hæmatoxylin gave good results, especially for the fibrillar elements after the above fixing solution and after also Mann's fluid.

A short account of the cell types in the spinal ganglia of the rabbit and of the dog and the changes produced by resection of the brachial plexus is very fully recorded. The following briefly recounts the author's results in the case of the latter animal.



There are five cell types. 1. Large clear cells, with granuli-form chromophile elements scattered almost equally throughout the protoplasm with a slight increase towards the cell periphery. The nucleus is large and clear and there is a definite perinuclear and peripheral clear zone. (2) Large and medium-sized cells with very fine chromophile elements, which are larger, however, at the periphery of the cell. Other characteristics as in type 1. (3) Small dark cells with very fine chromophile elements, which are larger around the nucleus with which they are in contact. The fundamental substance and nucleus are diffusely coloured. (4) Small and medium-sized clear cells with chromophile elements somewhat large and small in number. The nucleus is clear and separated from the chromophile substance by an achromatic zone. (5) Large clear cells with elongated chromophile elements lying concentrically around the nucleus in parallel planes. The nucleus is always excentric.

For the resection experiments ten healthy dogs were used. The brachial plexus was cut across, and part of each trunk excised. The animals were then killed on the 4th, 7th, 10th, 15th, 20th, 40th, 60th, 80th, 120th, 240th days. The 6th, 7th, 8th cervical and 1st dorsal ganglia were examined.

The first two cell types showed the same kind of alteration and were only distinguishable therefore in the initial stages.

In type 1 the chromophile material becomes very powdery at first in the centre, but the change quickly spreads to the periphery. In type 2 the peripheral chromophile elements are more resistive, and the finer internal elements degenerate from without inwards. Then the peripheral elements degenerate and it becomes impossible to separate type 2 from type 1.

The nucleus now passes to the periphery of the cell, and attached to its aspect which looks towards the centre of the cell, there is a crescentic chromophile mass. Should the nucleus remain central there are two paranuclear chromatic masses, one on each side. The process of alteration reaches its maximum on the 15th day, but examples can still be found on the 60th day. Repair can begin on or after the 15th day. The nucleus becomes rounded, and the paranuclear mass dissolves. The cell gradually fills with chromophile material, spreading from the nucleus to other regions. The nucleus then returns to the centre. After 80 days, types 1 and 2 can be distinguished. The cell repair is a purely morphological one, the cell never attaining to its original dimensions.

The small dark cells are somewhat rapidly altered. After four days the reaction has reached its height. The cytoplasm is pale, especially in the centre, the nucleus is peripheric with a small chromophile band on its central aspect. (The large varieties of

the type alter more slowly.) After ten days repair begins. In some the chromophile band attached to the nucleus breaks up, granules appear in the cell, especially near the nucleus, which remains peripheric. In others the nucleus passes rapidly to the centre with a rich zone of chromophile material around it. Sometimes the new chromophile mass is divided into two zones by a clear ring. Gradually the cell is filled with fine granules.

The small cells with large chromophile elements never show changes.

On the fourth day, in the vortico-se cell, the chromophile elements are condensed around the nucleus, while the peripheral parts are pale. On the tenth day the chromophile material around the nucleus may be limited to a few dark particles. In some cells the central elements are broken up and the nucleus peripheral. The peripheral elements then break up, and on the fortieth day this type cannot be distinguished from the first two.

When repair commences these cells re-acquire their distinctive characteristics.

On the sixtieth day there is formed a central mass, while the more distinct and elongated elements are situated at the periphery.

On the eightieth day the cells are more definitely vortico-se, and on the one hundred and twentieth day many cells are normal.

Here and there cells undergo complete degeneration and become surrounded and penetrated by capsular nuclei (fifteen to forty days), and as a result of their destruction one finds degenerated fibres in the posterior columns of the cord. Vacuolar degeneration is rare.

Cytometric data.—Many measurements are given in this part of the work of the cell bodies, nuclei, and nucleoli of the various cell types, but for these one must consult the original article.

The conclusions adduced from the data are as follows:—In the reactive phase, swelling takes place in the large clear cells and also in the small dark ones.

The swelling disappears rapidly and a diminution of the cell body takes its place. This occurs when the phase of reaction has reached its height. In the small dark cells there may be hypertrophy at an advanced stage of the reparative process, and Lugaro thinks that this is independent of the initial swelling, which is a fleeting phenomenon of the reactive phase.

This late hypertrophy does not occur in the large clear cells. Atrophy of a certain degree finally takes place in all the cells.

The nucleus of the large clear cells is seen to be diminished from the beginning. In the small dark cells it is seen to be increased in volume at the tenth day; it is then decreased and remains so. The nucleolus is increased in volume, and reaches its

maximum at the height of the reparative phase; it then diminishes, but is always larger than normal although the cell is atrophied. The nucleolar increase is most noticeable in the small dark cells.

On the following grounds Lugaro thinks the small dark cells have a greater resistive and reparative capacity than the large clear ones. The former react earlier, repair commences sooner, and they return to normal more quickly. The nucleus is increased in the reactive phase and the protoplasm hypertrophied in the reparative phase. They suffer a less degree of final atrophy, show more hypertrophy of the nucleolus, and amongst them there is less cell destruction. The latter react later and more slowly; the nucleolus is less hypertrophied; repair is very slow; there is no hypertrophy of the cell body in the reparative phase and never of the nucleus, for this is usually deformed and lessened in size; they show a greater degree of final atrophy, and amongst them there is more cell destruction.

The next part of the paper is taken up with the structure of the nerve cells in the plexiform ganglion of the vagus, and a detailed account of these cells is given as well as the changes induced in them after the experiments mentioned above.

The last part of the paper gives fully the changes induced in experiment 5.

In these two last sections the appearances both of the normal cells and of the phases of reaction and repair are carefully compared with those of the spinal ganglia in the first experiment, and for the difference one must consult the original article.

The following are the author's conclusions:—

The different morphological types of cells in the sensory ganglia must be considered as specifically distinct, both from an anatomical and probably also physiological standpoint.

The relative numerical proportion of different cell types is not accidental; it differs in different ganglia, but is constant in similar ganglia of individuals of the same species and even of different species.

There are no cells in the sensory ganglia, whose axone ramifies in the ganglion itself, *i.e.* independent of the peripheral nerve.

The various cell types react in a different manner, with different degrees of intensity and at different times.

In ganglia of different species the cells of a given type do not react in the same manner; in many cases, however, there is a considerable similarity in the appearance.

Between the homologous cells of the spinal ganglia and of the vagus ganglion of the same animal, there are some points of difference and some points of agreement in the type of reaction.

The small cells react and repair earliest. The earlier the reaction, the sooner repair sets in.

In the vagus ganglion reaction is earlier than in the spinal ganglia of the same animal.

A cell can remain at the height of reaction without progressing or regressing for a fairly long time.

Even when the nerve is prevented from reuniting, the process of repair always begins in all the ganglia and in all the cell types. But in some cells the reactive phase passes on to degeneration. Degeneration can take place at any phase of the alteration, and is greater in the vagus ganglia than in the spinal ganglia.

When the nerve is so injured that complete repair is impossible, and normal functional relations cannot be re-established, the corresponding cells, although in a phase of repair, show a distinct progressive atrophy.

Hypertrophy is exceptional and temporary. Hypertrophy of the nucleoli of cells whose fibre is injured, is an index probably of trophic hyper-activity on the part of the cell itself.

The classical type of reaction—central chromatolysis with peripheric position of the nucleus—is the most complete; the other types are more or less attenuated forms.

The cellular modifications produced by division of the nerves are indices of rejuvenescence plus exceptional trophic activity exercised by the cell during the period of regeneration of the nerve fibres.

The morphological characters of cells in the reactive phase are met with in cells undergoing development; they are also seen in various low forms of phylogenetic development. DAVID ORR.

**THE EXTERNAL FEATURES OF THE BRAIN OF A MICRO-  
(80) CEPHALIC IDIOT, SHOWING ABSENCE OF CORPUS  
CALLOSUM.** J. FROUDE FLASHMAN, *Reports from the Patho-  
logical Laboratory of the Lunacy Department (N.S.W.), Vol. i.,  
Part i., 1903.*

THIS is the preliminary account of a microcephalic brain presenting most exceptional features. The grey substance of the two cerebral hemispheres was in uninterrupted continuity, the corpora striata, the cortex of the frontal lobes and of the gyri fornicati (as far back as the situation of the pineal body) being completely fused.

This specimen reminds me somewhat of the brain of a Cyclops monster described by Cleland in 1878 (*Journal of Anatomy and Physiology*, vol. xii., plate 17), but the case under consideration belonged to a youth of seventeen years.

The only case of an adult brain at all resembling it of which we possess any record is, so far as I am aware, that described by Sir William Turner (*Journal of Anatomy and Physiology*, vol. xii., 1878; also *Brain*, vol. i., pp. 133, 134).

Turner's case was the brain of an epileptic imbecile of forty years, in which the convolutions in the mid-dorsal region were continued from one hemisphere into the other; but the frontal ends of the hemisphere were not united.

Several writers have attempted to explain such a monstrosity as being a persistence of the supposed primitive undivided condition of the fore-brain. But we now know, thanks largely to the work of Studnicka, that such a primitive undivided condition does not exist: the two cerebral hemispheres are developed independently, each from the dorsal part of the lateral wall of the neural tube. The apparently undivided fore-brain found in the Elasmobranch fishes and these human freaks are certainly the results of secondary fusion of two originally separate hemispheres.

G. ELLIOT SMITH.

#### ON THE ACTION OF VENOMS OF DIFFERENT SPECIES OF

##### (81) POISONOUS SNAKES ON THE NERVOUS SYSTEM.

GEORGE LAMB and WALTER K. HUNTER, *Lancet*, Jan. 2, 1904.

THIS paper is the first of a series which proposes dealing with the action of certain snake venoms on the nervous system, and it describes the histological appearances in the ganglion cells of six monkeys and three rats dying as the result of cobra venom intoxication. The changes in these ganglion cells varied with the length of time the animals lived after injection, and it seemed as if it were necessary that from two to three hours' time should elapse before chromatolysis would become apparent. Thus in three of the monkeys which died in  $2\frac{1}{2}$  minutes,  $3\frac{1}{2}$  minutes and 70 minutes respectively after injection, no definite appearance of degeneration could be made out. But in the monkey dying in 2 hours 30 minutes, and in another dying in 2 hours 45 minutes, there was fairly certain evidence of early chromatolytic changes. In the sixth monkey, dying 6 hours after injection, the degenerative changes were very well marked, and were fairly typical of an acute chromatolysis. One of the rats was kept alive, by the aid of an anti-venom serum, for 45 hours, with the result that the changes in its ganglion cells were most extreme, many of the cells being much broken up and vacuolated.

W. R. HUNTER.

#### INVASION OF THE CAUDA EQUINA BY A TUMOUR, WITH

##### (82) SUCCESSIVE DEMARICATION OF ALL THE SENSORY ROOT AREAS OF THE LOWER LIMBS. C. R. Box, *Lancet*,

Dec. 5, 1903, p. 1566.

A BOY of thirteen came to St Thomas's Hospital complaining of pain in the lower part of the back, the thighs and the knees, of nearly two months' duration. The only objective symptom was

loss of both knee-jerks. He remained under observation for seven months, and died later. No necropsy was obtained. Shortly after admission the plantar reflexes disappeared, and the calves in particular wasted rapidly. Anæsthesia corresponding to Head's first sacral and fifth lumbar roots developed, associated with retention of urine, some incontinence of feces, and loss of the right cremasteric reflex. An anæsthetic area corresponding to Head's fourth lumbar appeared on the inner side of the leg. A small anæsthetic area next appeared on the buttock above the gluteal fold, reaching the mid-line. Afterwards anæsthesia spread in both directions from the buttock outwards and downwards, and from the soles upwards. An incomplete band, defective behind, reaching from the top of the patella down over the upper third of the leg appeared to represent the third lumbar field. The upper limit of the second lumbar field crossed the thigh at junction of its upper and middle thirds. An area which appeared in the groin was taken to represent the last dorsal field. Between this and the preceding lay the first lumbar root area. The remaining sacral areas gradually developed, surrounding the area already mentioned on the buttock, and tailing off more and more down the back of the thigh and leg. Thus the fourth sacral strip reached half-way down the back of the thigh, and the third nearly to the back of the knee-joint. The second sacral area lay below the third, and probably embraced it above. The mutual relations of the sacral fields was like that shown by Starr, but their extent was different. The legs finally became completely anæsthetic and paralysed, with loss of faradic contractility. The first, second and third lumbar neural arches were removed, and a thickened, pulpy theca disclosed. No benefit accrued, and a large tumour developed in the neighbourhood of the scar.

The diagnosis was tumour of meninges or of bone commencing in the neighbourhood of the last lumbar and first sacral roots. Throughout the paralysis and loss of reflex was in advance of the appearance of the corresponding anæsthesia. All forms of sensation appeared equally and simultaneously affected. Hyperæsthesia preceded anæsthesia in most of the areas involved.

AUTHOR'S ABSTRACT.

### CLINICAL NEUROLOGY.

**A CASE OF GALLOPING PARALYSIS WITH SOME OBSERVATIONS ON THE SYMPTOMATOLOGY AND PATHOLOGICAL ANATOMY OF THE DISEASE.** WEBER, *Monatschr. für Psychiat. und Neurol.*, Dec. 1903.

THE patient was a man aged 34, highly gifted, but showed marked stigmata of degeneration. He had had syphilis sixteen years

previously. His illness began suddenly with acute excitement, and its course may be divided into three stages. First, there was the period of acute excitement with hallucinations, incoherence, grandiose ideas, etc.; the bodily symptoms were variable; then followed a period of remission during which almost all symptoms, mental and bodily, disappeared. This was succeeded by a period of acute depression, which lasted one month, and exhibited associative and psychomotor arrest, and the bodily symptoms became very marked. He died from cerebral exhaustion. At the post-mortem examination, the brain was found to be oedematous, hyperæmic, and there was a leptomeningitis commencing. Microscopically, marked changes were seen in the vessels, there were recent perivascular collections of nuclei, and there was also overgrowth of the neuroglia. Very little change was seen in the nerve cells and fibres.

During the first two periods, the diagnosis was not certain. The bodily symptoms were irregular, and the psychic symptoms closely resembled those met with in cases of acute insanity. But it is unusual to get such a rapid and complete remission, and the following depression was much more intense than is generally seen in the reactive stage of that condition. In the third period, however, the motor symptoms became much more typical, and these, taken into consideration with the changes found in the brain on microscopical examination, confirm the opinion that we are dealing here with a case of general paralysis which lasted six months, or, in other words, with a case of galloping paralysis.

But the question arises as to whether the disease had lasted longer than this without giving rise to any trouble which would bring the patient under observation. The author, in connection with this point, goes more fully into the history of the patient, and he mentions that signs of degeneracy (an undue conceit, an overbearing manner, and outbursts of irritability) had been noticed since his schooldays; moreover, his breakdown occurred shortly after he had been advanced to a higher post which carried with it an increased responsibility, and the strain of this at last proved to be too much for him, although he was able to carry out his duties up to the time of his outburst. The author suggests that any peculiarities he may have exhibited were due to his original disposition, and states that they did not increase up to the time of his illness. Nor do the pathological findings imply that there had been a long-standing disease.

With regard to the pathology of this case, the author considers that the tissues first affected were those of the walls of the medium and small sized vessels, *i.e.* the mesodermal tissues of the brain, while the ectodermal tissues were very little attacked, and that at a later period.

In these vessels he found the intima intact, but with its nuclei a little enlarged.

In the muscular coat, however, were many nuclei lying between the muscular fibres, and even the elastic coat was in some places broken up. These nuclei, he states, were nuclei of the connective tissue (*Bindegewebskerne*), and not plasma cells. Only in a more inflammatory condition are these last seen.

Around these affected vessels, the overgrowth of the neuroglia, and the degeneration in the nerve cells were more marked than elsewhere; but he considers both of these changes to have been secondary to the condition of the vascular walls.

He also mentions that cases of general paralysis do occur in which the degenerative process attacks the nervous and neuroglial tissues first, and to a large extent spares the vessels; but these are rare.

R. G. ROWS.

#### **PARTIAL PARALYSIS OF THE TONGUE AND LIPS IN A GIRL**

(84) **AGED 14, PROBABLY DUE TO ANOMALY OF THE MEDULLA.** VARIOT, *Bull. de la Soc. de Pédiat.*, No. 5, 1903.

In this patient, while the size and general appearance of the tongue were in no way abnormal, its movements, like those of the orbicularis oris, were materially impaired. The girl did not begin to speak until her sixth year; before that age her attempts to do so were quite inarticulate. There was constant dribbling of saliva until she was ten years old. At present her power of articulation is considerably interfered with, the majority of her words being pronounced badly, particularly those containing *d*, *t*, and *s* sounds, in which the tip of the tongue has to be applied to the teeth. She cannot protrude the tongue from the mouth, nor can she sweep its point round the dental arches, so that in eating she has either to use her finger to move the bolus of food after chewing it, or to swallow it without mastication at all. Though she can close her lips, the feebleness of the orbicularis is such as to make whistling, pouting, etc., impossible. The girl is otherwise healthy.

The condition must be due to a congenital lesion in the domain of the hypoglossal and facial nerves, analogous to that described by Möbius and Heubner.

J. S. FOWLER.

#### **CONTRIBUTION TO THE LOCALISATION OF HEMICHOREA.**

(85) **AUFSCHLAGER**, *Ztschr. f. klin. Med.*, Bd. 51, H. 3, 1904.

The writer first defines Hemichorea, in which the one-sided movements are rapid and associated with intention, and differentiates it from Hemithetosis, in which the movements are slow and constant. He states that these two conditions, however, belong together, because many intermediate cases are recorded, and



because hemichorea may develop into the other. This type of hemichorea is, however, to be distinguished pathogenetically from *Chorea minor* (St Vitus' Dance).

Charcot in 1885 suggested that in hemichorea and hemiathetosis a lesion of the posterior limb of the internal capsule is responsible, and he found in three autopsies that the lesion was situated in the hinder ends of the optic thalamus, caudate nucleus, and foot of the corona radiata. His hypothesis was that the choreiform movements depended upon the involvement of an undiscovered bundle of fibres of peculiar motor function, running along with the known paths in the corona.

Kahler and Pick (1879), on the other hand, held that the condition was due to irritation of the pyramidal fibres where they lie between optic thalamus and lenticular nucleus, and stated that in these they had repeatedly found a descending degeneration. Greiff in 1883 published two cases which strengthened their view.

The writer gives the clinical and post-mortem record of a woman who died of phthisis, at the age of 43, after having suffered 15 years from hemichorea of sudden onset, affecting the left arm and leg. There was practically no affection of sensation. The important part of the autopsy, as concerned the hemichorea, was the presence of an old fissure-like cyst, 3 cm. long, situated mainly in the hinder end of the right optic thalamus, and, at its posterior end, reaching into the white substance of the occipital lobe just beneath the ependyma of the lateral ventricle. A diagram of the position of the cyst is given.

JOHN D. COMRIE.

#### PERMANENT DYSARTHRETIC TROUBLE IN AN EPILEPTIC (86) OCHILD. DECROLY, *Journ. de Neurol.*, Jan. 5, 1904.

THE writer records a case in which a child of four years, and healthy parentage, developed epileptic seizures suddenly during the decline of an attack of whooping-cough. The first seizure appeared at the end of a paroxysm of coughing, and was in all respects a typical attack of *grand mal*. As time went on the attacks were repeated more and more frequently, especially at night, up to even fifteen times in one night, so that natural sleep was impossible. Gradually a physical and mental deterioration set in, and above all the child's speech became quite changed, questions being answered with effort and articulation being difficult. The child's vital functions were good and the reflexes normal. The treatment adopted was a mixed one of iodide, bromide, mercury, and warm bathing, under which, in some months, the fits passed off and speech slightly improved. The latter has, however, remained permanently stuttering, soft, and like the speech of general paralysis.

JOHN D. COMRIE.

**EMPHYSEMA OF THE SKIN AFTER AN EPILEPTIC ATTACK.**(87) RANSOHOFF, *Neurolog. Centralbl.*, Jan. 16, 1904.

THE occurrence of subcutaneous emphysema as a result of obstruction to forced expiration in child-bed, whooping-cough, etc., has been described by several writers, and Kloots has gathered forty cases from the literature of the subject. The writer does not find any case recorded as due to epilepsy, and gives a case where it occurred on one occasion in an epileptic. He suggests that the emphysema arises, not from interstitial emphysema of the lungs spreading by the peribronchial and mediastinal cellular tissue, as is generally supposed, but from a tear in the mucous membrane of the trachea.

JOHN D. COMRIE.

**(88) TIC OF THE EYES.** HENRY MEIGE, Paris, 1903, pp. 23.

IN this paper the writer first gives the points of distinction—as regards pathogenesis, clinical symptoms and prognosis—between the two kinds of convulsive movements, tic and spasm: these terms are used in the sense employed by Brissaud.

He then considers the various forms of tics of the eyes, dividing them into two groups: I. Tic of the Eyelids; II. Tic of the Globes.

I. Tic of the lids is the most frequent of all forms of tic; it may be of various kinds: (1) *Tic de nictitation*, slight and rapid movements of the upper eyelid; (2) *tic de clignotement*, much more common, all the fibres of the orbicularis being clonically contracted; (3) *tic de clignement*, a tonic form of the last, the eye being kept half closed; (4) *tic d'écarquillement*, clonic or tonic, with excessive opening of the palpebral fissure. Reference is also made to several cases affected with tic, in which the position of one or both upper eyelids suggested a *ptosis* of organic origin; it was, however, only a "way of holding the eyelids," at first deliberately adopted for a definite end, either to protect the eye against bright light or to express the state of lassitude of which the patient complained, but later, by repetition, becoming a habit and occurring involuntarily and unconsciously. As regards the causes of palpebral tics, most frequently they are started by some external irritation (a foreign body in the eye, conjunctivitis); but they may be caused by a repeated and strong visual impression, e.g. bright light, or by troubles of vision, such as those connected with errors of refraction. The chief points of distinction between tic of the eyelids (*blepharo-tic*) and spasm of the eyelids (*blepharo-spasm*) are these:—(1) true spasm is generally unilateral: tic is most often bilateral; (2) the characters of the convulsive movements are different: in spasm the movement begins in one part of a muscle, usually at the free border of the lower lid, and gradually spreads so as to affect different fasciculi of the same muscle, and may then

extend to other muscles; in tic the whole muscle at once comes into play, or, if only a part of a muscle contracts, it is because those fibres have a different function from the other parts; (3) palpebral spasm is only exceptionally accompanied by movement of the eyes, while this is very common in palpebral tic, in virtue of functional association, and for the same reason rotation of the head often occurs in tic of the eyeballs.

II. Tic of the eyeballs, affecting (a) extrinsic, (b) intrinsic muscles.

(a) Convulsive movements of the eyes, clonic and tonic, occur, which should be considered as tic: they may affect one eye or both, and they may exist independently of palpebral tic. These movements often occur at the same moment as the closure of the eyelids. A lateral movement of the eye may accompany a movement of the head in the same direction, as in one case cited. If the movements are of sufficient frequency, one sees a sort of nystagmus; he believes it proved that nystagmus occurs as a purely functional condition, and he refers to one published case of tic, and gives the notes of another in which nystagmus was marked, with no sign of organic disease—*nystagmiform tic*.

A tonic form of tic of these extrinsic muscles may be shown by a fixed position of the eyes, giving a wild or simply absent look. The same cause may explain certain forms of strabismus, common in infants, not due to any trouble of vision or anomaly of the ocular muscles, and curable solely by strict watching. It is not rare to find in the subjects of tic some difficulty in looking down: this may be due to tic of the elevators of the eye.

(b) Meige believes that the intrinsic muscles of the eye are also subject to tic. There are certainly functional troubles of accommodation, e.g. "professional spasm" seen in microscopists, opticians, etc. "One can conceive of the existence of a *tic of accommodation*," and he refers to one case of a subject of tic, with a stammer, in whom attacks of micropsia, lasting some minutes, are believed to be due to tonic tic of the muscles of accommodation. Perhaps, in other cases, megalopsia may have the same mode of origin, and the visual troubles, irregular and unaccountable, which occur in certain cases of tic, may be due to a clonic form of the tic of accommodation, with intermittent variations in the curvature of the lens.

Meige also suggests that the muscles of the iris may suffer from tic, e.g. that there may be a *hippiform tic*.

The paper concludes with some remarks on prognosis and treatment. The treatment is that of tic in general, viz., methodical exercises of the muscles involved. The patient should be taught to control the involuntary movements (*discipline d'immobilité*), and at the same time he must learn to execute, at command,

regular, slow, methodical movements (*discipline du mouvement*). These exercises should be done at first under the eye of the physician, but they should be repeated by the patient at home, and Meige strongly urges the advantage of these home exercises being performed before a mirror.

The prognosis is good. In the majority of cases a cure is obtained, and this is specially true of young patients. In infants, a parent who will pay enough attention and exercise enough firmness, can almost always prevent the development of tic if the symptoms are noted at the start. In adults, ocular tics are more obstinate, but we should always reckon on great improvement and even cure. Less favourable are the cases of old people, in whom the tics are the signs of commencing mental decay, but even in these one may hope for good result; it depends very greatly on the mental state.

ASHLEY W. MACKINTOSH.

**FACIAL SPASM: ITS DISTINCTIVE CHARACTERS.** HENRY (89) MEIGE, *Rev. Neurolog.*, No. 20, Oct. 30, 1903.

MEIGE agrees with Brissaud in insisting on the importance of distinguishing between facial spasm and facial tic. He believes that the diagnosis can be made on purely objective clinical grounds, and in the present paper he describes the clinical features of "facial spasm" in its different degrees of severity. Notes are given of nine cases. In one of these the spasm affected first one side of the face, disappeared, and then appeared later on the other side. Meige does not consider the aetiology or pathogenesis of facial spasm; he simply says that it is due to irritation, transitory or permanent, at some point in the reflex facial arc.

The chief clinical points of diagnostic value, as serving to differentiate facial spasm from other forms of convulsive movements affecting the face, notably tic, are these :—

(1) The spasm begins locally, and spreads more or less gradually, so as progressively to involve more and more muscles; usually it starts as a slight quivering at the free border of the lower eyelid, and thence gradually spreads so as to affect the whole of that eyelid, and, later, the upper eyelid; the spasm may stop here, but more generally it invades progressively the whole of the muscles of one-half of the face; it disappears in the same way, often rapidly, but not suddenly. There is no sudden appearance and disappearance of the complete series of phenomena, as in tic.

(2) Even in the severest degree of facial spasm, when there appears to be a permanent contracture of all the muscles of one side of the face, the contracture is never quite uniform; here and there portions of some muscles are relaxed, while other portions

are in a violent state of spasm; several muscles may be affected by the spasm *en masse*, but in others there are only fibrillary or fascicular contractions. In tic all the fibres of the affected muscles are equally involved.

(3) The affected part of the face is quite devoid of every known form of emotional expression; the muscular contractions show no trace of co-ordination for a definite act, *e.g.* mimetic, as is seen in tic.

(4) The phenomena are strictly limited to the region innervated by the facial nerve. There is never, as in tic, any implication of other muscles functionally associated with those of the face. (All the muscles supplied by a facial nerve are not necessarily involved; sometimes transient contractions are seen on the other side of the face.)

(5) No voluntary effort of any kind, and no psychical influence—except in rare cases—has the slightest effect on the convulsive movements. Hence facial spasm is but little amenable to the modes of treatment which are so successful in true tics.

(6) The spasm may appear or go on during sleep.

(7) The spasm is not accompanied by pain; there is none of the intolerable agony which characterises the affection known erroneously as tic douloureux of the face.

(8) The spasm obstinately resists all forms of treatment.

Meige refers to the three cases of facial spasm which have been recorded by Bernhardt, Newmark and H. Frenkel, who are inclined to regard them as examples of a special form of facial spasm, in view of the fact that the tonic spasm is combined with constant fibrillary movements in some of the affected muscles. Meige, however, is inclined to believe that these fibrillary movements form an integral part of the symptomatology of facial spasm, and that their presence does not constitute a special form of the condition.

He also considers the possibility of the simulation of facial spasm by hysteria, and he gives notes of one case (Observation X.) in which the diagnosis between these two conditions was doubtful. These cases are rare. Hysteria can scarcely reproduce all the clinical characteristics of facial spasm. One must, however, remember the possibility of a combination of the two conditions.

A. W. MACKINTOSH.

## TWO BROTHERS WITH SYMMETRICAL DISEASE OF THE

### (90) MACULA COMMENCING AT THE AGE OF FOURTEEN.

RAYNER D. BATTEN, *Trans. Ophthal. Soc.*, Vol. xvii, 1897, p. 48.

Two cases are recorded. In the first, a boy aged 14 years, there was failure of vision, so that he could only see  $\frac{1}{2}$  with either eye.

In both eyes at the macula there was a dark, pear-shaped patch of a red colour, dotted over with fine points of retinal pigment. Surrounding this macula there was an area of retinal disturbance dotted over with ill-defined yellowish-white dots. Visual fields showed no contraction, and there was no definite scotoma, though slight confusion of colour vision in a very small central area.

The second case was the elder brother of the above, aged 21 years. His vision had begun to fail when 14 years old. The changes were more advanced than in his brother's case. There was some fine choroiditis and retinal change occupying the whole of the macula region, forming an ill-defined area with a small central deposit of fine dotted pigment.

The following points are noted :—

(1) The failure of vision occurred at about the same age (viz. 14) in the two brothers. The onset was gradual.

(2) The changes were symmetrical in each case.

(3) The condition of the fundus in the elder brother probably represented a later stage of the condition now commencing in the younger brother's eye.

(4) There was a definite history of syphilis in the parents, and from the family history the patients were probably the subjects of hereditary syphilis, though they show no definite sign of that disease.

FREDERICK E. BATTEN.

#### **CEREBRAL DEGENERATION WITH SYMMETRICAL CHANGES**

##### **(91) IN THE MACULÆ IN TWO MEMBERS OF A FAMILY.**

F. E. BATTEN, *Trans. Ophthal. Soc.*, Vol. xxiii., 1903, p. 386.

Two cases are reported. The first was that of a girl aged 7—the fifth of a family of seven children. The first was a boy aged 15, healthy; the second, a girl aged 13, now in an asylum; the third, a boy, died of convulsions when  $2\frac{1}{2}$  years old; the fourth, a boy aged 9, healthy; the fifth, a girl aged 7, the first case described; the sixth, a boy aged 5, healthy; the seventh a girl aged  $1\frac{1}{2}$ .

About twelve months ago the child became spiteful at school and had violent attacks of temper, and about this time her sight began to fail. The physical condition of the child was good.

On ophthalmoscopic examination the discs were slightly pale, but not markedly atrophic. There were peppered pigmentary changes all over the retina. At each macula there was a reddish-black spot, larger and more defined in the left than in the right eye, about one-third of the size of the optic disc in diameter. The shape was irregular and not round, and the margin was not sharply defined. The region immediately surrounding the dark spot was paler than the rest of the fundus and more atrophic looking. The vessels were small (note by Mr W. T. Lister).

The elder child, the second in the family, complained of headache when four years old, and shortly after that it was noticed that her sight began to fail and her mental state deteriorated. When 9 years old she had a fit, and on two subsequent occasions had a recurrence of these fits.

The disc was pale, the edges fairly distinct, the vessels were small, there was considerable pigmentation in the region of the yellow spot (Mr Marcus Gunn's note).

The child's mental condition rapidly deteriorated and she went to the asylum. . . .

Both children are at present in the asylum, and are regarded by the authorities as "general paralytics." The elder child now shows some motor weakness; the younger child none.

AUTHOR'S ABSTRACT.

**PSEUDO - PARALYTIC MYASTHENIA GRAVIS AND PREG-**  
(92) **NANCY.** RUDOLF KOHN, *Prag. med. Wochensh.*, May 14, 1903.

KOHN adds another case of myasthenia gravis and pregnancy to the few already recorded, and reviews the literature on this subject.

His patient, twenty-seven years of age, a v-para, was seen at the fourth month of pregnancy. She had felt very weak for about fourteen months, ever since the birth of her last child. Any slight exertion, even walking, produced attacks of giddiness and faintness, and after exertion if she attempted to swallow any liquid, unless she swallowed it very slowly, the fluid regurgitated through the nose. After speaking for any time she had the feeling that she must speak through her nose. She exhibited all the characteristic symptoms of the disease, and the "myasthenic reaction" was present in the muscles, especially in the biceps. She had always been a healthy woman, and there was no history of alcohol or syphilis. On two occasions previously—ten years ago and six years ago—she had had attacks of fainting somewhat similar to those which she now complained of. During these attacks, which lasted over an hour, she said she was quite unconscious, and that she bit her tongue and lip. On examination no atrophy of the muscles was found, and the reaction of degeneration was not present. The field of vision was unimpaired, and the retinae were normal. There was no paralysis of the palate.

The patient was kept in bed for five days, and then the "myasthenic reaction" could not be obtained. She returned home and continued to improve, and the pregnancy ended normally. Two days later, however, she became very weak, the pulse was very feeble, and the breathing became difficult and stertorous in character. She died the same day. Kohn recapitulates the leading

features of the case, and says there can be no doubt that it was one of myasthenia gravis. Referring to the two unobserved attacks of fainting, with prolonged unconsciousness and biting of the tongue, Kohn considers that they were probably epileptic fits. While there is no reference in literature to the association of myasthenia gravis with epilepsy, Oppenheim mentions a case where a brother of the patient was an epileptic.

The pregnancy had a definite influence upon the course of myasthenia gravis in Kohn's case—development of the disease after delivery, ending very rapidly in death. In one previous case the patient died during pregnancy; and in another (Sinkler's), all the symptoms appeared suddenly after delivery. Goldflam had seen a case of myasthenia gravis, where on two occasions after delivery relapses occurred, and he had also noted the sudden appearance of almost all the symptoms at the end of the period of lactation.

The prognosis of myasthenia gravis is made worse by conception, and it would seem to be worth while to consider the question of the induction of premature labour in some cases. The prognosis as regards the child is favourable. OLIPHANT NICHOLSON.

#### RECURRING PAINS; OR, LES DOULEURS D'HABITUDE.

(93) BRISSAUD, *Progrès Méd.*, Jan. 9, 1904.

In a short but most instructive paper, Professor Brissaud analyses critically those cases of recurring pains, the nature, intensity, even the seat of which are of only secondary importance: the essential element is that they return at a given day or hour without any apparent external influences; or at any moment, yet associated with some circumstance or other at once invariable and insignificant. Thus one of his cases was a woman aged 26 years, who was awakened every night at two o'clock by an agonising cramp in the calf of the left leg, which lasted almost exactly for an hour. Another, aged 45, had suffered for twenty years from excruciating pains in the neck, face, arms and back, which occurred with mathematical regularity every morning at 11 and every evening at 7. The author further quotes from an old writer of the 16th century the following passage: "for three years and seven months every Monday and almost at the same hour, a Benedictine monk was tormented by a migraine of extreme violence affecting the right temple, which lasted from twenty-eight to thirty hours."

To begin with, neurasthenia (in the ordinary sense) and hysteria are carefully excluded. The first step in the analysis is the recognition of the *agonising* character of the pains, which, joined to their *periodicity*, indicates that one has really to deal



with an hallucination. A further characteristic is their *irresistibility*, which is admitted generally to be an essential element in the *idée fixe*. In these cases in which the crises are nocturnal, it is obviously not the pain which awakens the patient, though because it follows immediately he is apt to believe it is; rather it is because the patient is awake that he feels the pain; he has contracted the habit of waking at a given moment, and as probably at first this waking was really because of the pain, *now* he is so much under the influence of the "fixed idea" that this "paroxysmal nocturnal anxiety" is *sufficient to reproduce the pain* when he does awake.

Another curious case is cited where a bank clerk, aged 27, fatigued from overwork, noticed, or thought he noticed, that the cause of his fatigue lay in the reading or writing of long numbers. He then thought it was the reading or writing of long words. He continued, however, to attempt this writing and reading, with the consequence that he began to have pains in the neck and back, shooting down to the testicles. Now the mere effort to read a word of more than a certain length produces at once an altogether sickening pain in the testicles. The author brings this case into line with the others by pointing out that it is essentially an hallucination; that a relation of cause and effect purely imaginary has become a pathological cerebral "habit."

The remark of Madame de Sévigné, "I cannot bear either the sight or even the *imagination* of a precipice"; the popular phraseology which says, "I have only got to think of it to have cold sweats"; the shudder which passes through one at the mere *memory* of the sound of a chalk pencil scraping on a black-board; these are only minor degrees of the same hallucinatory phenomenon.

S. A. KINNIER WILSON.

**ON THE DIFFERENTIAL DIAGNOSIS OF KORSAKOFF'S**  
(94) **DISEASE.** SERGE SOUKHANOFF et ANDRÉ BOUTENKO,  
*Journ. de Neurol.*, Dec. 5, 1903.

THE authors regard Korsakoff's disease as an autonomous morbid process in which mental symptoms are associated with phenomena of polyneuritis. Alcohol is the commonest cause, but typhoid fever, jaundice, etc., may produce it. In mild cases one may find relative amnesia as regards present and recent events, while the patient retains his personality and vivacity of spirits. Careful examination may be needed to determine the affection of memory, while neuritis symptoms are marked. In bad cases fallacious or pseudo-memories may appear, while in still worse cases the talk has a fantastic character. The amnesia concerns present and recent

events only, and hardly at all those in the more distant past. There are, during the course of the disease, frequent oscillations in the degree of amnesia present, depending chiefly on the physical state of patient for the time being. Some systematising delirium is often present. The progress of the disease, for better or worse, is slow. Complete recovery, if possible, is rare.

K. d. is produced by autointoxication: the expression of a special reaction of the organism in the presence of an extreme amount of toxic element. In the case of alcoholism, there is not merely the reaction of the organism in the form of d. t., but also a secondary reaction tending to paralyse the defensive powers. Post-typhoidal K. d. most frequently develops in connection with a relapse. So also in cases from jaundice, prolonged action of toxin is necessary.

The authors proceed to consider K. d. with reference to other morbid conditions. The following points are selected:—

(1) *Alcoholic amnesia*.—While previous to K.'s writings, affection of memory in alcoholism had been noted, K. was the first who directed attention to the special amnesia associated with polyneuritis. In K. d. the amnesia element is more accentuated and may be accompanied by false memories. The onset is more acute and the patient more reasonable. In ordinary alcoholic amnesia the symptom is never so isolated, but develops gradually and more or less parallel with other changes in psychic activity. (2) *Primary confusional insanity*.—The authors do not share the view that K. d. is simply a form of this; the marked mental confusion observed in K. d. being only one of the transitory manifestations and not an essential element. (3) *Senile dementia*.—While the resemblance between this and K. d. may be close, the amnesia in the former is slowly progressive and accompanied by other symptoms of senile loss. Mental processes in S. d. take on a monotonous character, while in K. d. all the stores of psychic representation remain practically intact. (4) *Arterio-sclerosis*.—In these cases there may be a comparatively rapid development of the mental troubles, their more progressive character, enfeeblement of memory, and general loss in psychic spheres, serve as differentiating features. (5) Cases of *apoplexy* may resemble K. d.; but one must bear in mind that a "shock" sometimes occurs at the commencement of true K. d. (6) In *cerebral tumour* the mental symptoms may stimulate K. d., there being in some an affection of memory out of proportion to that of the other mental functions, but the more or less marked phenomena of dementia in such are a guide. In a series of cases, the majority of those which had isolated affection of memory, were cases of tumour of the hypophysis. Does such a lesion favour the development of K. d.? Pseudo-memories are rare in cerebral tumour, where careful ex-

amination nearly always reveals loss in mental activity. (7) *Cerebral syphilis*.—In such cases general mental slowness, weakness in power of combination, habitual changeableness of temper, irritability and loss of interest, are among the differentiating symptoms. Confabulation and pseudo-memories are less marked than in K. d. (8) *General paralysis*.—Many cases of K. d. have been mistaken for this. Cranial nerves and pupil reflexes may be affected in K. d., and disturbances of speech and ideas of grandeur may occur. G. p. may resemble K. d., amnesia for recent events and pseudo-memories being present. The lesion in g. p. is a more profound one. There is not so marked a difference in the memory of recent and remote events as in K. d., but the individuality is changed. (9) *Amnesia*, sometimes retrogressive, is met with in certain cases of *traumatic neuroses*, *hysteria*, *epilepsy*, and after *poisoning by carbonic acid*. (10) *Polioencephalitis hæmorrhageia superior*.—The authors record a case of K. d. in which the *sectio* revealed large hæmorrhagic softenings in the region of the frontal and parietal lobes. It is possible that such are connected with modifications in vessel walls produced by the same special toxin which has to do with the psychic and nerve affections of K. d. Such post-mortem results do not, however, militate against the diagnosis of K. d. It is to be remembered that in the cases which have been adduced in proof of the assertion that K. d. may occur without polyneuritis, no microscopical examination of nerves has been made, and also that in some cases of K. d. neuritic symptoms are less marked clinically and rapidly disappear. The data do not really contradict the view that K. d. is characterised by the co-existence of special psychic troubles and of polyneuritis.

But every case of psychic disturbance which also shows signs of polyneuritis must not be looked on as one of K. d. It is not uncommon in chronic alcoholism to have symptoms due to lesion of peripheral nerves accompanied by passing symptoms of d. t. The characteristic of K. d. is the combination of special mental and nerve symptoms, both being due to one toxin in the circulation.

A. HILL BUCHAN.

**A FACIAL REFLEX.** A. FUCHS, *Neurolog. Centralbl.*, 1904, (95) p. 15.

A **REFLEX** contraction of the muscles at one or both angles of the mouth, but chiefly of the zygomaticus and levator labii superioris, is obtained by gently pressing on the one eyeball.

It was present in about half the normal cases examined, and is always absent in facial paralysis, but it is not noted if it can be used to distinguish between central and peripheral palsies.

GORDON HOLMES.

**CONCERNING THE SKIN REFLEXES OF THE LOWER EXTREMITIES (IN PARTICULAR THE BABINSKI REFLEX).**  
(96) **TREMITIES (IN PARTICULAR THE BABINSKI REFLEX).**  
S. GOLDFLAM, *Neurolog. Centralbl.*, 1903, pp. 1109, 1137.

WHEN testing the plantar reflex in doubtful cases, the writer recommends the physician to engage the patient's attention all the while in conversation, in order to eliminate as far as possible any superadded voluntary movements. And the result of the first stimulation of the sole is the one to be taken, inasmuch as later results are more likely to be complicated by defensive or by voluntary movements. In about 14 per cent. of healthy adults the plantar reflex is absent, and in about the same proportion of patients suffering from non-organic neuroses. There is no necessary connection between the briskness of the reflex movement and the intensity of the sensation of tickling in the sole.

Goldflam describes the various minor varieties of plantar reflex occurring in healthy adults. He records his experience of the plantar reflex in organic affections of the pyramidal tract and emphasises the fact that the Babinski phenomenon may often be elicited immediately after the onset of a hemiplegia, before the clinical signs of localised paralysis have made their appearance.

The Babinski reflex is a more constant and more reliable phenomenon in hemiplegia than is ankle-clonus. Thus in 43 cases of hemiplegia, the extensor plantar reflex was present in every case, whilst foot-clonus was observed only in 28. In hysteria and neurasthenia, where a pseudo-ankle-clonus occasionally is a source of difficulty, the presence of a normal plantar reflex is of great significance.

In cases of monoparesis due to encephalitis, even where the lower limb was implicated, he has found absence both of foot-clonus and of the Babinski reflex.

For the production of the Babinski phenomenon, it is immaterial whether the lesion be situated in the cortex, white substance of the hemisphere, or mid-brain. He has found it present in one case of Gubler's palsy (anterior part of pons), where ankle-clonus was absent; in one case of Weber's palsy (crus cerebri), both ankle-clonus and Babinski phenomenon were absent, whilst in another instance of lesion of the crus it was uncertain, owing to the persistent extended posture of the hallux and the co-existence of post-hemiplegic choreiform movements. In this latter case the Babinski phenomenon appeared after several months on the non-paralysed side, accompanied by ankle-clonus.

In a case of internal hydrocephalus with hemiplegia, in which the muscles of the other side were also spastic, the Babinski reflex was present on both sides, without ankle-clonus, though all the tendon reflexes were brisker than normal. In a case of infantile

diplegia, though the knee-jerks were increased, neither ankle-clonus nor Babinski phenomenon were present.

In some instances he has observed a crossed Babinski reflex, *i.e.* extension of the hallux on stimulation of the sole of the opposite, unaffected foot.

Goldflam's observations in cases of cerebral tumours, multiple sclerosis, syringomyelia, myelitis, etc., agree in the main with the results obtained by most other observers and need not here be repeated. With one exception, in all his cases of functional neuroses the plantar reflex, if obtained at all, was invariably flexor in type. This exception was in a man of 42, neurasthenic, with mitral incompetence and bleeding piles, in whom the Babinski phenomenon was present on both sides, also a slight ankle-clonus and increased knee-jerks. Goldflam attributes these appearances to secondary anæmic changes in the cord, analogous to the degenerations common in pernicious anæmia.

Observations were also made on the plantar reflex during sleep in healthy children between the ages of 6 and 16 years (no adults were available in hospital). In a large proportion of these, about one half, he obtained an extensor response, whose constancy was proportional to the soundness of the sleeping state. In these sleeping children he also observed certain of the tendon reflexes, particularly the ankle-jerk, sometimes also the knee-jerk, though the latter was more difficult to elicit without awakening the patient. In 30 out of 40, one or both of these reflexes could be obtained, contrary to the experience of Rosenbach, who states that the knee-jerks are absent during deep sleep.

The plantar reflex was also observed during ether anæsthesia (a preliminary administration of morphia was given in each case before commencing the administration of ether), and it was found that the reflex disappears generally very shortly after the appearance of anæsthesia. Meanwhile the tendon reflexes become exaggerated and ankle-clonus appears and can be elicited throughout the period of anæsthesia. The plantar reflex remains absent for hours after the anæsthesia has passed off. Similar results were obtained in chloroform cases, but in a certain proportion of the latter, the deep reflexes also disappeared during anæsthesia. Neither in ether nor in chloroform narcosis was the Babinski reflex observed.

These facts indicate that the centres for the two plantar reflexes and for the tendon reflexes are widely different in situation. The normal plantar reflex is referable to the cerebral cortex, and not to the spinal cord. Munk's experiments on dogs and monkeys also corroborate this view. The normal flexor response, then, is a cortical reflex, the Babinski extensor phenomenon a spinal one. If the periphereo-cortical reflex path be interrupted by an organic

lesion, or if the activity of the cortical centres be interrupted, as in ordinary sleep, the normal flexor response disappears and the spinal plantar reflex, extensor in type, becomes evident.

PURVES STEWART.

## PSYCHIATRY.

### FAMILY AMAUROTIC IDIOCY AND ITS DIAGNOSIS. A.

(97) MÜLBAYER, *München. med. Wochenschr.*, No. 45, 1903.

IN the hitherto recorded cases of this disease, the typical appearance of the fundus has always been present, and all the reported cases have been in Jewish children.

The author describes two cases, a brother and a sister, in whom cerebral degeneration and loss of sight were the prominent symptoms, but in whom the characteristic fundus change was not present. The parents were Germans, and perfectly healthy, and lived under healthy conditions.

The first child, a boy, was born five years after marriage, and until five months old was considered to be healthy. The mother then noticed that the child no longer followed her movements with his eyes. When the child was a year old he was seen by an ophthalmic surgeon, who stated that the child was blind.

When the child was  $3\frac{1}{2}$  years old he came again under observation. In the course of the years which had passed the child had steadily degenerated. The child presented all the features of an idiot. The eyes were in constant movement. The pupils were dilated and did not react to light.

On ophthalmoscopic examination the following appearance was noticed in the left eye:—The papilla oval in perpendicular direction, of a dirty grey colour, and the vessels were very small. The macula region was about equal to the diameter of two papillæ. It was of a pale red colour, and was surrounded by a circle of granular pigment of a black colour. There was no central red spot. The rest of the fundus showed an irregular pigmentation.

In the right eye a similar condition was present.

The second child, a girl  $1\frac{1}{2}$  years old, had also become blind. The child could stand, but could not walk. She was very irritable, and laughed without cause. The globe of the eye was in constant movement.

The ophthalmoscopic examination was as follows:—In the left eye the papilla was round, of a reddish grey colour, and the vessels were very small. No marked change could be seen in the macular region. In the inner and lower quadrant towards the periphery there was a series of five parallel wavy lines of black pigment. The rest of the fundus had a granular appearance.

The right eye showed the same atrophy of the nerve, and the same granular appearance of the fundus as the left, but no gross change was present.

In the first of these children there was advanced grey atrophy of the optic nerve, with complete amaurosis and changes of the macular region.

In the second child there was grey atrophy of the optic disc, but no change at the macula. There was probably some appreciation of light. The boy was an idiot; the girl was probably so. The points in which these cases do not agree with the "type" are the long duration of the disease in the boy, the character of the fundus change, and the onset of the latter.

In the reported cases the macula disease has been the primary change, and the optic atrophy followed; whereas in the boy the appearance suggests that the optic atrophy was primary and the fundus change followed, and this was certainly so in the case of the girl.

The writer places these cases into the "family amaurotic idiocy" type, but they differ from the "type" in that the fundus change is not typical.

FREDERICK E. BATTEN.

**ON THE QUESTION OF COMBINED PSYCHOSES.** ROBERT GAUPP.  
(98) *Centralbl. f. Nervenheilk. u. Psychiat.*, Dec. 1903.

It has been recognised for many years, at least on the Continent, that patients, suffering from chronic mental disease, may, at the same time, suffer from an acute form of an entirely different nature, and hence give rise to the clinical picture of a "combined psychosis." For example, an imbecile may develop some form of acquired psychosis, a paranoiac may develop delirium tremens, or an epileptic may develop any form of chronic mental disease. Hebephrenia may supervene in a patient who is already weak-minded ("Pfropfhebephrenie"). Ferenczi has recognised such combinations as hysteria and paranoia, alcoholism and general paralysis, paranoia and general paralysis, imbecility and alcoholism, etc.

Such combinations as the above are, as a rule, fairly obvious; but, having regard to Kraepelin's classification, which is so rapidly gaining ground, and realising the difficulty of "pigeon-holing" all cases according to this classification, the author of the present paper wishes to go a step further. He suggests, for example, that the characteristics of katatonia and hebephrenia may occur in the same patient, that the childish excitement of the hebephreniac may occasionally occur in the "unproductive mania" of manic-depressive insanity, and that the flight of ideas characteristic of manic-depressive insanity may occur in the course of a true paranoia. These and many other instances which are given in the

paper suggest that the clinical picture of many cases will be rendered clearer by a more frequent recognition of states of combined psychoses.

W. H. B. STODDART.

**THE PREVAILING CONCEPTION OF DEGENERACY AND DE-  
(99) GENERATE, WITH A PLEA FOR INTRODUCING THE  
SUPPLEMENTARY TERMS DEVIATION AND DEVIATE.**

G. L. WALTON, *Boston Med. and Surg. Journ.*, Vol. cl., Jan. 21, 1904.

THE term degenerate is now of interest, not only to the anthropologist, but to the medical and general public, and the time has come to question whether it is adequate with no synonym, except for its worst significance, to meet all requirements of scientific classification and discussion, to say nothing of general use. The term superior degenerate has led to confusion; applied in its academic sense only to individuals whose higher qualities are affected, the tendency has crept in to include under it individuals with minor peculiarities whether physical or mental.

The standard definitions of degeneration, in its anthropological sense, imply reduction to a lower type, especially with respect to moral qualities; its synonyms are limited to such words as depravity, demoralisation, prostitution, vitiation, blight, rottenness and pollution. But medically speaking, degeneration is defined as a condition in which there is marked deviation from the average normal, and its stigmata include such insignificant deviations as difference in colour of the two eyes. It does not follow because many degenerates show many deviations from the average normal, that every such deviation is degenerate; when we include all deviations under this term we have drifted into the unscientific position of recording *facts* in such a way as to involve an *opinion*. Even if it should eventually be proved (which is highly improbable) that every deviation means degeneration, nothing would have been lost by following a logical plan in establishing that proposition. According to the present nomenclature, congenital absence of vermiform appendix would be classed as degenerative if some individual should be born so happily constituted. The etiology of so-called degenerative stigmata has been made to include syphilis, alcoholism, depravation and allied conditions in the ancestry (Nordau), and again, toxic infections affecting the developing embryo (Vaschide and Vurpas), signs of atavism and evolutionary spurts, shown *e.g.* by unusual development of Broca's convolution in an individual possessing unusual command of language (Lombroso). It would seem that the downward is not the only direction in which deviation may be found. Even Lombroso in classifying genius as a degenerative psychosis, does



not claim that every genius is, as a whole, necessarily deteriorate, in fact, he warns against the exaggeration of deducing degeneration from single facts, and yet he has no other name for the most trivial of these facts than signs of degeneration.

If one term must be chosen to include all varieties of deviation with their varied etiology, it is unfortunate that a name should be chosen which has of necessity a sinister significance, and is by no means uniformly appropriate. In the light of such considerations the use of the terms "deviation" and "deviate" will not only serve accurately to classify the phenomena, but may even contribute toward a better understanding of the complex conditions with which we are concerned.

It would certainly answer every purpose, for example, to substitute *deviation* in the statistics which state that 4 per cent. of normal individuals have five or more signs of degeneration, as compared with 27.4 per cent. of delinquents. The use of the word delinquent in these statistics shows that a mild designation may be used to cover a severe type, for delinquent is here made to include the most hardened criminal, and yet in the same statistics the word degeneration is obliged to include the most insignificant signs of deviation.

It is not intended, however, to supplant the word degeneration, but to limit its application to stigmata, to individuals, to families, or to races showing unquestioned downward tendency, and it is especially intended to replace, as far as practicable, that self-contradictory and unnecessarily opprobrious designation, superior degenerate.

AUTHOR'S ABSTRACT.

## TREATMENT.

**DIETETIC BROMIDE TREATMENT OF EPILEPSY.** MEYER,  
(100) *Berl. klin. Wochenschr.*, Nov. 16, 1903, p. 1049.

RICHER and Toulouse in 1889 recommended that the administration of bromide in epilepsy be supplemented by exclusion of salt from the food. Including the natural salt of the food, the maximum allowed was 2 grammes daily. Meyer treated four cases, each suffering from almost daily fits, by this method as modified by Balint, in which the latter excluded flesh from the dietary and substituted bread (named Bromopan) salted with Sod. Bromid.

The diet was :—

$\frac{1}{2}$ – $\frac{3}{4}$  litre cocoa or coffee.

450 grammes Bromopan (1 gramme bromid in 150 gr. bread).

$1\frac{1}{2}$  litres milk.

3 eggs (unsalted).

Fruit.

In each case a period of observation, lasting four weeks, preceded the administration of the diet and medicine. Three patients were observed for two, three, and five months respectively, and in all the treatment was greatly superior to ordinary bromide medication. The attacks were fewer and less severe, improvement occurring after three to ten days.

The fourth patient was observed for twelve months with an interruption of fourteen days.

During four weeks preceding treatment the fits were severe and occurred almost daily. The treatment was arranged in five periods.

Period I. Bromopan diet (3 grammes Na. Br. per diem). Four weeks. Increase of fits for ten days and then twenty-one days with none. Last six days, 1.5 gr. Na. Br. daily. A few days after stopping treatment, fits resumed former type.

Period II. Ordinary diet and administration of 3.0 to 8 grammes Na. Br. gradually increased and then decreasing till finally stopped. Seven to eight weeks. When 8 grammes were administered (*i.e.* after four weeks), the fits stopped for twenty days. Later under 3 grammes bromide the fits were almost of the original severity, and two days after cessation they were frequent. Gradually the type changed and free intervals (three to eight days) occurred, followed by a day with one to three fits.

Period III. Ordinary diet with administration of Opocerebrin (Poehl), 0.3 grammes thrice daily. Six weeks. No change.

Period IV. Bromopan (3 grammes Na. Br. daily). Eight weeks. Dieted for two and three-quarter months more, gradually diminishing bromide. After fourteen days there came a period of two and a half months without fits. Ten days after stopping treatment there were six slight fits on one day. Patient left hospital for a fortnight and returned as bad as ever.

Period V. Five weeks without medication. Very few fits occurred, and he was allowed home, and remained without treatment for three and a half months. There were few fits of the old type, and the mental state was better until the sudden onset of epileptic stupor without fits.

Meyer's patients bore the treatment well, and all improved in weight. Reported unfavourable results can be explained by inadequate surveillance, and diminution in weight occurs only when the patient refuses the prescribed amount of food. Bromide acne and bromide intoxication are more easily induced. That the beneficial results may be entirely due to the regulation of the life in an institution is disproved by the observations made before special treatment was begun. The author strongly recommends a trial for this treatment.

J. EASON.

## Reviews

**FATIGUE.** A. MOSSO, Professor of Physiology in the University of Turin. Translated by MARGARET DRUMMOND, M.A., and W. B. DRUMMOND, M.B., C.M., F.R.C.P.E. London: Swan, Sonnenschein & Co., Ltd.; New York: E. P. Putnam's Sons, 1904.

At the present time, when so much attention is devoted to physical deterioration, to over-pressure in schools, and to the increase of nervous disease and insanity, there is need for an authoritative account of what is known of the pathology of fatigue. Such an account no one is better qualified to provide than Professor Mosso, the eminent Turin physiologist; and in the book before us he has dealt succinctly, but in a spirit both practical and scientific, with the main features of the subject. The opening chapters deal with the general and special characteristics of fatigue as exhibited in the muscular and nervous systems, and as studied by aid of the ergograph. The writer does not profess to be able to explain with certainty the ultimate cause of fatigue, but he is convinced that it is essentially a toxic phenomenon, a poisoning of the nerve centres by the products of their own, and of muscular activity. He is no believer in the independence of muscular and nervous fatigue. "The exhaustion of energy," he says (p. 243), "is general; and all the magazines of energy can be drained by the exaggeration of any activity whatever of the organism. The conclusion to which we are led by my experiments is that there exists only one kind of fatigue, namely, nervous fatigue; this is the preponderating phenomenon, and muscular fatigue also is at bottom an exhaustion of the nervous system." The remaining chapters deal with the most important part of the book—that which is concerned with intellectual fatigue. He points out here the important physiological fact that it is not only the brain, but also the muscles, which are exhausted by hard intellectual work; and draws from that fact conclusions of the greatest practical importance. Professor Mosso is the happy possessor of a lucid and allusive style of writing, and no higher praise can be given to the work of the translators than to say that in their hands these qualities have been in no degree impaired. The book is one which can be read with the greatest pleasure as well as profit, and we cordially commend its careful study, not only to medical men, but to all who are interested in educational matters and to the great army of intellectual workers in general.

R. HUTCHISON.

**THE "JOURNAL DE PSYCHOLOGIE NORMALE ET PATHOLOGIQUE."** Directeurs: Dr PIERRE JANET; Dr GEORGES DUMAS. Première Année—No. 1. Felix Alcan, 108 Boulevard Saint-Germain, Paris.

THE present year has already seen the birth of a *British Journal of Psychology*, and the appearance of another French journal devoted to the interests of psychology is an indication of the zeal with which the science is being pursued on the other side of the Channel. It is intended to publish the journal every two months, and the annual volume will contain about 600 pages. A special feature will be made of analyses of recent literature, so that the journal may "become a sort of *Centralblatt* for all who are interested in the study of psychology, normal and pathological." The first number of the journal contains 112 pages, of which 75 are devoted to abstracts. These number 84 and are classified under suitable headings. Of them 39 are from French papers or books; 24 from English and American; 8 from German; 8 from Italian; and the remainder include Russian and Hungarian, so that a pretty wide field is covered.

There are four original articles. The first, by Professor Ribot, deals with the "Value of Questionnaires in Psychology," and is rather severely critical. The conclusion reached is that the method can only be an auxiliary in the study of psychology, if one accords to criticism the important rôle which it deserves. This is a double one: criticism of evidence; criticism of the use made of it. We wish this article had been carried a little further. The "questionnaire" has become extremely popular in recent years, particularly in America, and it is high time the scope and the limitations of the method, and the fallacies dependent upon it, should be fully discussed by a competent psychologist. Perhaps Professor Ribot will return to the subject.

The second communication is a note by Professor Flournoy relative to a spiritualistic seance.

Professor Grasset, in the third article, discusses the sensation of "deja-vu," the feeling, that is, that something one has just seen for the first time has been seen before, one knows not where or when. The various theories which have been brought forward to explain this feeling are criticised, and Professor Grasset enunciates a theory of his own which does not seem to differ materially from that which Lemaitre propounded in a recent paper (see p. 226).

The last of the original papers, by Professor Raymond and Janet, deals with the phenomena of loss of personality (dépersonnalisation) and possession as illustrated by a particular patient. The authors explain their reasons for regarding the symptoms they describe, not as somnambulistic hysteria, but as a crisis of

obsession in a psychasthenic, and point out how the diagnosis influences the prognosis and treatment.

The next number of the journal is to contain papers upon "Head's Zones and their importance in Psychiatry" (Pick); "A Peculiarity of the Mental State of Morphinomaniacs" (Brissaud); "The Influence of Alimentation on Character" (Houssay); and others.

The *Journal de Psychologie* has made a good beginning and the programme of papers promised for 1904 excites our interest and expectation. We wish our contemporary all success.

W. B. DRUMMOND.

**DIE MESSUNG DER PUPILLENGROSSE, UND ZEITBESTIMMUNG DER LICHTREACTION DER PUPILLEN. ALFRED FUCHS. 1904.**

THIS work consists of 134 pages, of which 50 consist of tables of observations, and 10 of bibliography. The author advocates the use of mechanical apparatus in recording pupillometric observations, and after a brief historical review of the efforts in this direction, he describes the various forms of pupillometer in use in recent years, such as Morton's, Haab's and Schnabl's, each dependent on the comparison of the observed pupil with a graduated series of circles of known diameter. With the use of such apparatus it is not difficult to appreciate changes in the pupil diameter of 0.1 mm., but it is impossible with them to record the psychical reactions of the pupil and those of convergence. A further improvement was made by the "strabometer" of Lawrence with tangential scale markings on a half-dial attached to a handle. With Schloesser's instrument it is possible to measure very rapidly. It consists of a narrow flat measure, engraved with two lines meeting at a point at a narrow angle, and divided by fine lines. This is repeated in larger size on the other side, so that variations of  $\frac{1}{4}$  mm. can be recognised between  $\frac{1}{4}$  and 15 mm. in width. After describing Schirmer's projection method of pupillometry, he refers to the more recent and accurate photographic method of Bois-Raymond by magnesium flash-light. By this means, by varying the strength of the light to which the pupil is exposed between the flashes, a curve of contraction from its fullest size in darkness down to its greatest contraction of 4 mm. is obtained. Straub found that the normal pupil width constantly became smaller with increasing age till the fiftieth year, and that in myopia the pupils were larger, and in hypermetropia smaller than in emmetropia. The latent period of the pupil reaction is also measured, and found to be slightly longer in the opposite

eye in consensual reaction. Detailed lists of pupil reactions in different diseases are given, and separate paragraphs are devoted to observations on cases of various forms of insanity, hysteria, neurasthenia, epilepsy, alcoholism, tabes and general paralysis, gross nervous diseases, tetany, disseminated sclerosis, and morphinomania. After a profuse bibliography, six plates are added, giving illustrations of the method of photographic record of pupil changes in various diseases. The whole work is ponderous in the extreme, and is a typical German production of the poorer quality, rich in bibliography and other spadework, and strikingly barren in original research or new discovery.

WILFRED HARRIS.

## Bibliography.

### ANATOMY

- Histological Studies on Cerebral Localisation. *Brit. Med. Journ.*, Feb. 6, 1904, p. 328.  
 ZUCKERKANDL. Zur vergleichenden Anatomie des Hinterhauptlappens. Deuticke, Wien, 1904, M.3.  
 GÉRARD. Les voies optiques extra-cérébrales. *Journ. de l'anat. et physiol.*, jan.-fév. 1904, p. 22.  
 DORENDORF and SCHULTZ. Ueber die centripetale Leitung des Nervus recurrens. *Arch. f. Laryngol. u. Rhinol.*, Bd. 15, H. 2, 1904, S. 217.  
 ONODL. Das Verhältnis der hintersten Siebbeinzelle zu den Nervi optici. *Arch. f. Laryngol. u. Rhinol.*, Bd. 15, H. 2, 1904, S. 259.  
 RETHI. Die sekretorischen Nerven des weichen Gaumens. *Wien. med. Presse*, Jan. 31 und Feb. 7, 1904, S. 214.  
 FERRONCITO. Sur les terminaisons nerveuses dans les muscles striés. *Gazz. med. ital.*, Dec. 24, 1903, p. 511.  
 WARNCKE. Zur Darstellung der Axencylinderfibrillen in den markhaltigen Fasern des Centralnervensystems nebst Bemerkungen zur Histologie des Centralnervensystems nebst Bemerkungen zur Histologie des Axencylinders im Allgemeinen. *Arch. f. Psychiat. u. Nervenh.*, Bd. 38, H. 1, 1904, S. 156.

### COMPARATIVE ANATOMY—

- KINGSBURY. Columella Auris and Nervus Facialis in the Urodela. *Journ. Comp. Neurol.*, vol. xiii., No. 4, 1903, p. 313.  
 ROTHMANN. Ueber das Verhalten der Arteria cerebri anterior beim Affen, Anthropoiden und Menschen. *Arch. f. Psychiat. u. Nervenh.*, Bd. 38, H. 1, 1904, S. 278.

### METHODS—

- STRONG. Notes on the Technique of Weigert's Method for staining Medullated Nerve Fibers. *Journ. Comp. Neurol.*, vol. xiii., No. 4, 1903, p. 291.

### PHYSIOLOGY

- JUDSON HERRICK. The Doctrine of Nerve Components and Some of its Applications. *Journ. Comp. Neurol.*, vol. xiii., No. 4, 1903, p. 301.  
 C. POLI. Sulla funzione centrifuga delle radici posteriori della midolla spinale. *Ann. di Freniat.*, vol. xiii., f. 4, p. 320.  
 GIUSEPPE PAGANO. Una prova dell' esistenza dei nervi trofici. *Riv. di patol. nerv. e ment.*, vol. ix., f. 1, 1904, p. 17.  
 ERNST MAI. Ueber gekreuzte Lähmung des Kältesinnes. Beitrag zur Physiologie der Hautsinnesbahnen. *Arch. f. Psychiat. u. Nervenh.*, Bd. 38, H. 1, 1904, S. 182.

- JENKINS and CARLSON. The Rate of the Nervous Impulse in the Ventral Nerve-Cord of Certain Worms. *Journ. Comp. Neurol.*, vol. xiii, No. 4, 1903, p. 259.
- LUIGI LUGIATO. Il tempo di contrazione muscolare latente studiato coll' ergografo nell' uomo. *Riv. di patol. nerv. e ment.*, vol. ix., f. 1, 1904, p. 1.
- ZACCARIA TREVES. Sui rapporti che corrono tra la curva del sollevamento ergografico e la curva della contrazione muscolare volontaria. *Riv. di patol. nerv. e ment.*, vol. ix., f. 1, 1904, p. 14.
- BEARD. A Morphological Continuity of Germ-Cells as the Basis of Heredity and Variation (cont.). *Rev. of Neurol. and Psychiat.*, Feb. 1904, p. 114.
- CARLSON. Beiträge zur Physiologie des Nervensystems der Schlangen. *Arch. f. d. ges. Physiol.*, Bd. 101, H. 1 u. 2, 1904, S. 23.
- GILDEMEISTER. Ueber ein mechanisches-Modell eines Nervmuskelpreparates. *Arch. f. d. ges. Physiol.*, Bd. 101, H. 1 u. 2, 1904, S. 52.

## PSYCHOLOGY

- JOSIAH ROYCE. Outlines of Psychology: An Elementary Treatise with Some Practical Applications. Macmillan Co., New York, 1903.
- KOWALEWSKI. Studien zur Psychologie des Pessimismus. Bergmann, Wiesbaden, 1904, M.2.80.
- RIBOT. De la valeur des questionnaires en psychologie. *Journ. de Psychol.*, jan.-fév. 1904, p. 1.
- HEYMANS. Untersuchungen über psychische Hemmung (iii.). *Ztschr. f. Psychol.*, Bd. 34, H. 1, 1904, S. 15.
- GROOS. Das Seelenleben des Kindes. Reuther und Reichard, Berlin, 1904, 3a. 6d.
- STRONG. Leib und Seele. *Ztschr. f. Psychol.*, Bd. 34, H. 1, 1904, S. 48.
- RAGNER VOGT. Ueber den Begriff der "Anregung" bzw. "Perseveration der kortikalen Vorgänge." *Centralbl. f. Nervenheilk. u. Psychiat.*, Jan. 15, 1904, S. 29.
- GRASSET. La sensation du "déjà vu." Sensation du "déjà entendu"; du "déjà éprouvé"; illusion de "fausse reconnaissance." *Journ. de Psychol.*, jan.-fév. 1904, p. 17.
- ALFRED BORSCHKE. Untersuchungen über die Herabsetzung der Sehschärfe durch Blendung. *Ztschr. f. Psychol.*, Bd. 34, H. 1, 1904, S. 1.
- MARX LOBSEIN. Über Farbenkenntnis bei Schulkindern. *Ztschr. f. Psychol.*, Bd. 34, H. 1, 1904, S. 29.
- FLOURNOY. Note sur une communication typtologique. *Journ. de Psychol.*, jan.-fév. 1904, p. 11.
- KRONTHAL. Die Beziehungen des Nervensystems zur Psyche. *Neurol. Centralbl.*, Feb. 15, 1904, S. 154.
- A. PICK. On Contrary Actions. *Journ. Nerv. and Ment. Dis.*, Jan. 1904, p. 1.
- DEL GRECO. Intorno alla scienza dell' individualità psicologiche umane (la psico-antropologia ed il suo metodo). *Il Manicomio*, vol. xix., No. 3, p. 305.
- RAYMOND et JANET. Dépersonnalisation et possession chez un psychasthénique. *Journ. de Psychol.*, jan.-fév. 1904, p. 28.
- LOUGUE. Etude médico-psychologique sur Dostoiewsky. Considérations sur les états morbides liés au génie (thèse). Storek et Cie, Paris, 1904.
- ALSBERG. Erbliche Entartung bedingt durch soziale Einflüsse. Fisher & Co., Cassel, 1904, M.0.80.
- TH. ZIEHEN. Einige Bemerkungen zur Anwendung der Methode der richtigen und falschen Fälle bei psychologischen Untersuchungen. *Monatsschr. f. Psychiat. u. Neurol.*, Bd. 15, H. 1, 1904, S. 64.
- A. PICK. Zur Pathologie des Ich-Bewusstseins. Studie aus der allgemeinen Psychopathologie. *Arch. f. Psychiat. u. Nervenh.*, Bd. 38, H. 1, 1904, S. 22.

## PATHOLOGY

- SABRAZÈS, MURATET et BONNES. Cellule nerveuse libre dans le liquide céphalo-rachidien, dans un cas de syphilis médullaire probable. *Arch. gén. de méd.*, Jan. 26, 1904, p. 218.
- ERNST SIEFERT. Ueber die Hirnmetastasen des sogenannten Deciduoma malignum. *Arch. f. Psychiat. u. Nervenh.*, Bd. 38, H. 1, 1904, S. 1.

- DANA and HASTINGS. On Cytodiagnosis in Nervous Diseases. With a Description of the Technique of Cell Diagnosis. *Med. Rec.*, Jan. 23, 1904, p. 121.
- MEYER. Ueber cytdiagnostische Untersuchung des Liquor cerebrospinalis. *Berl. klin. Wchnschr.*, Feb. 1, 1904.
- FRAENKEL. Lymphocytosis of the Cerebro-spinal Fluid. *Med. Rec.*, Jan. 23, 1904, p. 125.
- MINGAZZINI. Experimentelle und pathologisch-anatomische Untersuchungen über den Verlauf einiger Bahnen des Centralnervensystems. *Monatschr. f. Psychiat. u. Neurol.*, Bd. 38, H. 1, 1904, S. 52.

## CLINICAL NEUROLOGY AND PSYCHIATRY

### GENERAL—

- The National Hospital for the Paralyzed and Epileptic and Organised Scientific Research. Leading Article. *Lancet*, Jan. 30, 1904, p. 313.
- HOLST. Erfahrungen aus einem vierzigjährigen neurologischen Praxis. Enke, Stuttgart, 1904, M.1.60.

### MUSCLES—

- MEINERTZ. Zur Casuistik der Myokymie. *Neurol. Centralbl.*, Feb. 1, 1904, S. 101.
- HERMAN LUNDBORG. Ist Unverricht's sogen. familiäre Myoklonie eine klinische Entität, welche in der Nosology berechtigt ist? *Neurol. Centralbl.*, Feb. 15, 1904, S. 162.
- BRASCH. Dystrophia muscularis progressiva bei Mutter und Kind. *Berl. klin. Wchnschr.*, Jan. 18, 1904, S. 90.
- G. VON RITTER. Zur Kenntnis der progressiven spinalen Muskelatrophie in frühen Kindesalter. *Jahrb. f. Kinderh.*, Feb. 1904, S. 224.

### SPINAL CORD—

- TAYLOR and WATERMAN. Diffuse (combined) Degeneration of the Spinal Cord. *Boston Med. and Sur. Journ.*, Dec. 24, 1903, p. 695.
- COLLINS. A Case of Primary Sclerosis of the Posterior Columns, followed by Disseminated Softening of the Other White Columns. A Contribution to the Study of the Subacute Combined Degenerations of the Spinal Cord. *Rev. of Neurol. and Psychiat.*, Feb. 1904, p. 97.
- L. B. SCOTT. Detachment of the Odontoid Process of the Axis with Fracture of the Atlas; Continued Life without Symptoms of Injury to the Spinal Cord. *Brit. Med. Journ.*, Jan. 30, 1904, p. 247.
- Tabes.—HASSIN. Contribution to the Obersteiner-Redlich Theory of Tabes Dorsalis. *Med. Rec.*, Jan. 30, 1904, p. 168.
- FRANKLIN STAHL. The Association of Valvular Heart Disease with Tabes. *Am. Med.*, Philadelphia, Nov. 14, 1903.
- ERR. Syphilis and Tabes. *Berl. klin. Wchnschr.*, Jan. 18 und 25, 1904, S. 92.
- DALOUS. Les accidents syphilitiques pendant le tabes. *Rev. de Méd.*, Jan. 1904, p. 71.
- LESSER. Zur Aetiologie und Pathologie der Tabes, speciell ihr Verhältniss zur Syphilis. *Berl. klin. Wchnschr.*, Jan. 25, 1904, S. 80.
- ROSENBACH. Ist für die Entstehung der Tabes die Syphilis oder ein socialer Factor maassgebend? *Berl. klin. Wchnschr.*, Feb. 15, 1904, S. 174.
- Amyotrophic Lateral Sclerosis.—ALEX. M'PHEDRAN. Amyotrophic Lateral Sclerosis. *Am. Med.*, Philadelphia, Dec. 12, 1903.
- ALEX. M'PHEDRAN. Amyotrophic Lateral Sclerosis. *Canada Lancet*, Jan. 1904.

- Spinal Tumours.—M. VON SWITALSKI. Ueber wahre Neurome des Rückenmarks und ihre Pathogenese. *Polnisches Arch. f. biol. u. med. Wissensch.*, Bd. 2, H. 2, 1904, S. 158.

### BRAIN AND MEMBRANES—

- TANIGUCHI. Ein Fall von Distomumerkrankung des Gehirns mit dem Symptomencomplex von Jackson'scher Epilepsie, von Chorea und Athetose. *Arch. f. Psychiat. u. Nervenh.*, Bd. 38, H. 1, 1904, S. 100.
- Meningitis.—WARRINGTON. Case of Pneumococcal Meningitis and some Records of the Value of the Cytological Examination in Cases of Meningitis. *Rev. of Neurol. and Psychiat.*, Feb. 1904, p. 107.
- ELY and SNYDER. Contribution to the Clinical Study of Cerebrospinal Meningitis. *Am. Med.*, Philadelphia, Nov. 1903.



- LUTIER. Nouveaux procédés d'investigation dans le diagnostic des méningites tuberculeuses. Steinheil, Paris, 1904, 4 fr.
- AUCHE. Contribution à l'étude de la méningite cérébro-spinale diplo-pneumococcique de l'enfance. *Rev. mens. des mal. de l'enfance*, Jan. 1904, p. 20.
- JAMES GILROY. Notes on the Treatment of Tuberculous Meningitis. *Lancet*, Jan. 30, 1904, p. 295.
- Lumbar Puncture.**—MAX BLUMENTHAL. Seröse Meningitis und Lumbal-punction. *Arch. f. Kinderh.*, Bd. 38, H. 1 u. 2, 1904, S. 18.
- PERCHERON. Du diagnostic de la méningite tuberculeuse chez l'enfant. Valeur de la ponction lombaire. Steinheil, Paris, 1904, 3 fr.
- Hydrocephalus.**—SOUTHARD and ROBERTS. A Case of Chronic Internal Hydrocephalus in a Youth. *Journ. Nerv. and Ment. Dis.*, Feb. 1904, p. 73.
- Microcephalus.**—M. PROBST. Zur Lehre von der Microcephalie und Makro-gyrie. *Arch. f. Psychiat. u. Nervenh.*, Bd. 38, H. 1, 1904, S. 47.
- Memiplegia.**—ED. LONG. Un cas d'hémiplégie de cause cérébrale avec hémian-aesthésie persistante. *Rev. Neurol.*, fév. 15, 1904, p. 113.
- THEODORE DILLER. Hemiplegia, with Aphasia occurring during the course of Typhoid Fever in a Boy aged five years. *Med. Rec.*, Dec. 19, 1903, p. 991.
- KUTNER. Hemiplegia alternans superior (Störung der räumlichen Orientierung des Körpers). *Neurol. Centralbl.*, Feb. 15, 1904, S. 167.
- Bulbar Paralysis.**—HELENE FRIEDERIKE STELZNER. Ein atypischer Fall von Bulbärklähmung ohne anatomischen Befund. *Arch. f. Psychiat. u. Nervenh.*, Bd. 38, H. 1, 1904, S. 171.
- Intracranial Tumours.**—RAYMOND. Tumeur cérébrale; abolition des réflexes tendineux. *Arch. de Neurol.*, Jan. 1904, p. 1.
- BRISAUD et BACH. Fongus de la duremère. *Arch. gén. de méd.*, Jan. 26, 1904, p. 193.
- FRAENKEL and RAMSEY HUNT. Tumors of the Ponto-Medulla-Cerebellar Space. Acoustic Neuromata. (Central Neuro-fibromatose.) *Med. Rec.*, Dec. 26, 1903, p. 1001.
- NAGBOTTE. Sur la nature et la pathogénie des lésions radiculaires de la moelle qui accompagnent les tumeurs cérébrales. *Rev. Neurol.*, Jan. 15, 1904, p. 1.
- LESZYNSKY. Report of a Case of Brain Tumour involving the Right Lateral Ventricle. *Med. Rec.*, Jan. 30, 1904, p. 165.
- Cerebral Abscess.**—H. HORACE GRANT. Abscess of the Brain. *Louisville Monthly Journ. of Med. and Sur.*, Dec. 1903.
- General Paralysis.**—GAUPP. Die Prognose der progressiven Paralyse. *Deutsche med. Wchnschr.*, Jan. 21, 1904, S. 125.
- RAVIART et CAUDRON. Fréquence et évolution des lésions du fond de l'œil dans la paralysie générale. *Journ. de Neurol.*, fév. 5, 1904, p. 54.
- TOULOUSE et VURPAS. De la réaction pupillaire prolongée aux toxiques comme signe précoce de la paralysie générale. *Journ. de Neurol.*, fév. 5, 1904, p. 52.
- Brain Injury.**—ALEX. RAE. Head Injuries; their Diagnosis. *Brooklyn Med. Journ.*, Nov. 1903.
- CRISP ENGLISH. Hunterian Lectures on the After-Effects of Head Injuries. *Lancet*, Feb. 20, 1904, p. 485.
- BARTLETT and SCHWAB. A Case of Traumatic Injury to the Brain involving the Tongue Centre: Operation. *Interstate Med. Journ.*, Dec. 1903.
- LINDSAY. A Case of Perforating Wound of both Cerebral Hemispheres. *Journ. Am. Med. Ass.*, Jan. 23, 1904, p. 221.

#### GENERAL AND FUNCTIONAL DISEASES—

- CORNELIUS. Die Druck- oder Schmerzpunkte als Entstehungsursache der sogenannten funktionellen Nervenerkrankungen, ihre Entstehung und Behandlung. *Wien. med. Wchnschr.*, Jan. 16 und 23, 1904, S. 114.
- Paralysis Agitans.**—BURZIO. Ricerche sulla anatomia patologica del morbo di Parkinson. *Ann. di Freniat.*, vol. xiii, f. 4, p. 341.
- Epilepsy.**—W. B. FLETCHER. Masked Epilepsy. *Med. and Sur. Monitor*, Indianapolis, Nov. 15, 1903.
- MARCO-LEVI BIANCHINI. Épilepsie paranoïde. *Rev. Neurol.*, Jan. 15, 1904, p. 15.
- RAECKE. Das Verhalten der Sprache in epileptischen Verwirrtheitszuständen. *München. med. Wchnschr.*, Feb. 9, 1904, S. 256.
- LUNDBORG. Die progressive Myoklonin-Epilepsie. *Almqvist und Wiksell Upsala*, 1904, M. 4.50.

- Neurasthenia.**—JOSEPH N. STUDY. *Neurasthenia. Med. News*, Jan. 23, 1904, p. 159.
- CAPPELLETTI.** *La Nevrastenia*, con proemio del Prof. C. Bonfigli. Manuale Hoepli, Milano, 1904.
- Traumatic Neuroses.**—ERBEN. Ueber die gelaugigste Form der traumatischen Neurose. *Wien. med. Wchnschr.*, Jan. 30, 1904, S. 206.
- Hysteria.**—GANSER. Zur Lehre vom hysterischen Dämmerzustande. *Arch. f. Psychiat. u. Nervenl.*, Bd. 38, H. 1, 1904, S. 34.
- FREUND.** *Hysterie der Frau*. Simion, Berlin, 1904, M. 1.
- BARTH.** Manifestation of Traumatic Hysteria in the Organ of Hearing. *Arch. of Otol.*, vol. xxxii., No. 6, p. 443.
- THOMAS D. SAVILL.** A Clinical Lecture on Hysterical Skin Symptoms and Eruptions. *Lancet*, Jan. 30, 1904, p. 273.
- WOLLENBERG.** Die Hypochondria. Hölder, Wien, 1904, M. 2.
- Exophthalmic Goitre.**—ARNOLD SCHWYZER. Graves' Disease, with Report of Five Operated Cases. *St. Paul Med. Journ.*, Jan. 1904.
- Tetanus.**—BOUCHAUD. Un cas de tétanos céphalique avec diplégie faciale. *Journ. de Neurol.*, fév. 5, 1904, p. 41.
- HELLWIG.** Zur Serumtherapie des Tetanus. *Deutsche med. Wchnschr.*, Feb. 11, 1904, S. 235.
- Tetany.**—FILIA. La tétanie chez les enfants. *Rev. d'Hygiène et de med. infantiles*, T. ii., No. 6, p. 505.
- Tic.**—RUDLER et CHOMEL. Tics et stéréotypies de léchage chez l'homme et chez le cheval. *Nouv. Icon. de la Salpêtrière*, nov.-déc. 1903, p. 369.
- Acromegaly.**—JULIUS GRINKER. Acromegaly with Epilepsy. *Chicago Med. Rec.*, Dec. 15, 1903.
- Cretinism.**—BAYON. Beitrag zur Diagnose und Lehre vom Cretinismus. Stuber, Würzburg, 1904, M. 4.

#### MENTAL DISEASES—

- NEUMANN.** Versammlung südwestdeutscher Irrenärzte in Karlsruhe. *Monatschr. f. Psychiat. u. Neurol.*, Bd. 38, H. 1, 1904, S. 67.
- REDMOND.** Précis des Maladies Mentales. Rudeval, Paris, 1904, 4 fr.
- WERNICKE.** Outlines of Psychiatry in Clinical Lectures. *Alienist and Neurologist*, Nov. 1903.
- OTTO JULISBURGER.** Zur sozialen Bedeutung der Geisteskrankheiten, Berlin, 1903.
- C. B. BURR.** The Early Diagnosis of Mental Diseases. *Medicine*, Detroit, Nov. 1903.
- WALTON.** The Prevailing Conception of Degeneracy and Degenerate, with a Plea for Introducing the Supplementary Terms Deviation and Deviate. *Boston Med. and Sur. Journ.*, Jan. 21, 1904, p. 61.
- OTTO GROSS.** Ueber Bewusstseinszerfall. *Monatschr. f. Psychiat. u. Neurol.*, Bd. 38, H. 1, 1904, S. 45.
- HOWARD F. HANSELL.** Hallucinations of Vision. *Am. Med.*, Jan. 16, 1904.
- LOEWENFELD.** Die psychischen Zwangsvorstellungen. Bergmann, Wiesbaden, 1904, 14/.
- PFFERSDORFF.** Ueber symptomatische Zwangsvorstellungen. *Monatschr. f. Psychiat. u. Neurol.*, Bd. 38, H. 1, 1904, S. 20.
- ESPOSITO.** Sulle malattie mentali familiari (cont.). *Il Manicomio*, vol. xix., No. 3, p. 401.
- J. SIKORSKI.** Die russische psychopathische Literatur als Material zur Aufstellung einer neuen klinischen Form, der Idiophrenia paranoides. *Arch. f. Psychiat. u. Nervenl.*, Bd. 38, H. 1, 1904, S. 251.
- ADAM WIZEL.** Ein Fall von phänomenalem Rechenalent bei einem Imbecillen. *Arch. f. Psychiat. u. Nervenl.*, Bd. 38, H. 1, 1904, S. 122.
- PELLETIER.** L'association des idées dans la manie aiguë et dans la débilité mentale. Storck et Cie, Paris, 1904.
- SCHOTT.** Klinischer Beitrag zur Lehre von der chronischen Manie. *Monatschr. f. Psychiat. u. Neurol.*, Bd. 38, H. 1, 1904, S. 1.
- MOURATOFF.** Étude de la démence catatonique. *Arch. de Neurol.*, Jan. 1904, p. 14.
- DIEFENDORF.** Early Symptoms of Dementia Praecox. *Pediatrics*, Jan. 1904, p. 8.
- STRANSKY.** Zur Lehre von der Dementia praecox. *Centralbl. f. Nervenheilk. u. Psychiat.*, Jan. 15, 1904, S. 1.

- DARCANNE.** Contribution au diagnostic clinique de la démence précoce (thèse). Michalon, Paris, 1904, 4 fr.
- WILLIAM PICKETT.** Senile Dementia: a Clinical Study of Two Hundred Cases with Particular Regard to Types of the Disease. *Journ. Nerv. and Ment. Dis.*, Feb. 1904, p. 81.
- THOMSEN.** Aperçus et démonstrations sur la folie maniaque dépressive. *Journ. de Neurol.*, Jan. 20, 1904, p. 28.
- ERNEST W. H. BOOKER.** Some Notes on Melancholia and Mania, with Differential Diagnosis. *Memphis Med. Monthly*, Nov. 1903.
- P. PENTA.** Un pedofilio fellatore. *Riv. mens. di Psichiat. forense*, Anno vi, Num. 12, p. 477.
- OLIVA.** Due casi di inversione sessuale: Appunti clinici e medico-legali. *Ann. di Freniat.*, vol. xiii, f. 4, p. 289.
- HENRY MEIGE.** Documents complémentaires sur les possédés dans l'Art. *Nouv. Icon. de la Salpêtrière*, Nov.-Dec. 1903, p. 411.
- Criminal Psychiatry.**—**ANGIOLELLA.** Della criminalità nei pazzi. *Il Manicomio*, vol. xix., No. 8, p. 845.
- H. E. ALLISON.** Insanity in Penal Institutions and its Relation to Principles of Penology. *Albany Med. Ann.*, Dec. 1903.
- Forensic.**—**SANGER BROWN.** A Report of Three Medico-legal Cases involving the Diagnosis of Chronic Delusional Insanity. *Am. Journ. Med. Sc.*, Jan. 1904, p. 112.
- The Illegal Detention of Lunatics. Leading Article. *Lancet*, Jan. 30, 1904, p. 812.

#### ALCOHOL, ETC.—

- EDUARD HIRT.** Der Einfluss des Alkohols auf das Nerven- und Seelenleben. Bergmann, Wiesbaden, 1904, M.1.60
- SOUKHANOFF et WEDENSKY.** Délire alcoolique continu. *Nouv. Icon. de la Salpêtrière*, nov.-déc. 1903, p. 391.
- JOFFROY.** Délires systématisés spiritues. *Arch. gén. de méd.*, Jan. 28, 1904, p. 223.
- "Cures" for Alcoholism. Editorial, *Brit. Med. Journ.*, Feb. 6, 1904, p. 316.
- VAN VLEUTEN.** Ein Delirium im Anschluss an Hyoscinmissbrauch. *Centralbl. f. Nervenheilk. u. Psychiat.*, Jan. 15, 1904, S. 19.
- GUILLAIN et COURTELLEMENT.** Polynévrite sulpho-carbonée. *Rev. Neurol.*, fév. 15, 1904, p. 120.

#### CRANIAL NERVES—

- HOWELL T. PERSHING.** Optic Neuritis of Unknown Origin. *Journ. Nerv. and Ment. Dis.*, Jan. 1904, p. 29.
- BRISAUD et BRÉCY.** Neuromyéélite optique aiguë. *Rev. Neurol.*, Jan. 30, 1904, p. 49.
- ANTONELLI.** Les névrites optiques au cours des infections aiguës. Steinheil, Paris, 1904, 3 fr.
- DURANTE.** Névrome adipeux diffus du médian; résection; régénération autogène. *Nouv. Icon. de la Salpêtrière*, nov.-déc. 1903, p. 321.
- J. M. BALL.** Influence of Resection of the Cervical Sympathetic, in Optic Nerve Atrophy, Hydrophthalmos and Exophthalmic Goitre. *Journ. Am. Med. Ass.*, Jan. 30, 1904, p. 287.
- Facial Paralysis.**—**BIENFAIT.** Un cas particulier de paralysie faciale. *Journ. de Neurol.*, Jan. 20, 1904, p. 21.
- DUPUY-DUTEMPS et CESTAN.** Un phénomène palpébral constant dans la paralysie faciale périphérique. *Journ. de Neurol.*, fév. 5, 1904, p. 48.
- LENOBLE et AUBINEAU.** Paralysie alterne double incomplète limitée à la face. Faiblesse de tout le côté gauche du corps. Tubercules pédonculo-protubérantiels. *Rev. Neurol.*, Jan. 15, 1904, p. 12.
- VERGER.** Essai de classification de quelques névralgies faciales par les injections de cocaïne loco dolenti. *Rev. de Méd.*, Jan. 1904, p. 34.

#### REFLEXES—

- FRAENKEL and COLLINS.** Muscle Tonus and Tendon Phenomena. *Med. Rec.*, Dec. 12, 1903, p. 929.
- OPPENHEIM.** The Diagnostic Significance of Certain Reflexes and Tendon Phenomena. *Med. Rec.*, Jan. 2, 1904, p. 8.
- KNAPP and THOMAS.** The Reflexes in Long Distance Runners. A Study of the Influence of Fatigue upon Certain Reflexes. *Journ. Nerv. and Ment. Dis.*, Feb. 1904, p. 94.

- EMIL REDLICH. Ueber zwei neue Hautreflexe an den unteren Extremitäten. *Neurol. Centralbl.*, Feb. 1, 1904, S. 97.  
 BABINSKI. Sur la transformation du régime des réflexes cutanés dans les affections du système pyramidal. *Rev. Neurol.*, Jan. 30, 1904, p. 58.

#### MISCELLANEOUS SYMPTOMS—

- GEORGE M. GOULD. The History and Etiology of "Migraine." *Journ. Am. Med. Ass.*, Jan. 16, 1904, p. 168, 239.  
 JAMES SAWYER. Insomnia: its Causes and Cure. Cornish Bros., Birmingham, 1904, 1s. 6d.  
 PRAGER. Kopfschmerz und Schlaflosigkeit und deren Selbstbehandlung. *Orania-Verlag*, Oranienburg, 1904, M.1.  
 WILLIAM OSLER. On the Visceral Manifestations of the Erythema Group of Skin Diseases. *Am. Journ. Med. Sc.*, Jan. 1904, p. 1.  
 DELAGE. Fracture incomplète de l'olécrane et névrose traumatique. *Arch. gén. de méd.*, fév. 2, 1904, p. 279.  
 PIC et BONNAMOUR. Des troubles médullaires de l'artério-sclérose. *Rev. de Méd.*, Jan. 1904, p. 4.  
 BRISSAUD. Arthropathies syringomyéliques. *Arch. gén. de méd.*, déc. 29, 1903, p. 8281.  
 COMBE. Syndrome de Benedict inférieur. *Rev. mens. des mal. de l'enfance*, Jan. 1904, p. 1.  
 P. C. KNAPP. Bony Sensibility. *Journ. Nerv. and Ment. Dis.*, Jan. 1904, p. 25.  
 MINOR. Ueber die Localisation und klinische Bedeutung der sogen.: "Knochen-sensibilität" oder des Vibrationsgefühls. *Neurol. Centralbl.*, Feb. 15, 1904, S. 146.  
 DEJERINE et EGGER. Les troubles objectifs de la sensibilité dans l'acroparesthésie et leur topographie radulaire. *Rev. Neurol.*, Jan. 30, 1904, p. 54.  
 FINLAY. Delirium after Eye Operations. *Arch. of Ophthal.*, Jan. 1904, p. 5.  
 FUCHS. Die Messung der Pupillengröße und Zeitbestimmung der Lichtreaktion der Pupillen bei einzelnen Psychosen und Nervenkrankheiten. Deuticke, Wien, 1904, M.5.  
 COLLINS. Chronic Cyanosis of the Extremities Associated with Polycythæmia and Splenomegaly. *Med. Rec.*, Nov. 21, 1903.  
 MAURICE DIDE. Le pseudo-oedème catatonique. *Nouv. Icon. de la Salpêtrière*, nov.-déc. 1903, p. 347.  
**Aphasia.**—CHARLES L. DANA and JOSEPH FRAENKEL. A Case of Aphasia with Loss of Memory of Nouns (Sensory Anomia), with Autopsy. *Journ. Nerv. and Ment. Dis.*, Jan. 1904, p. 15.  
 KNAPP. Ein Fall von motorischer und sensorischer Aphasie. (Seelenblindheit und Seelentaubheit.) *Monatsschr. f. Psychiat. u. Neurol.*, Bd. 15, H. 1, 1904, S. 31.  
 POPOFF. Ueber amnestische Aphasie. *Neurol. Centralbl.*, Feb. 1, 1904, S. 106.  
 CHARLES H. HUGHES. Glossary of the Aphasias, Asymbolias and Alexias, with Comments. *Alienist and Neurologist*, Nov. 1903.  
**Amnesia.**—LACOMBE. Des principales formes d'amnésie et plus spécialement de l'amnésie traumatique envisagées au point de vue médico-légal. *Thèse de Paris*, 1903.  
**Alexia.**—PHILIP ZENNER. A Case of Alexia. *Journ. Nerv. and Ment. Dis.*, Feb. 1904, p. 89.  
**Dysantigraphia.**—GULBENK. Sur un cas de dysantigraphie. *Rev. Neurol.*, fév. 15, 1904, p. 123.

#### TREATMENT—

- J. F. WOODS. An Address on the Psychic Side of Therapeutics. *Lancet*, Feb. 20, 1904, p. 489.  
 EDWARD WALLACE LEE. Psychic Healing; or, properly speaking, the Treatment of Disease by Mental Influences. *New York Med. Journal*, Jan. 16, 1904, p. 97.  
 MEYER. Beitrag zur medicamentösen Behandlung von Neuralgien und Myalgien. *Berl. klin. Wchnschr.*, Feb. 8, 1904, S. 133.  
 WEDENSKY. Die Erregung, Hemmung, und Narkose. Hager, Bonn, 1904, 6a.  
**Electrical.**—FRAENKEL. Über chronische ankylosierende Wirbelsäulenversteifung. *Fortsch. a. d. Gebiete der Röntgenstrahlen*, Bd. 7, H. 2, 1904, S. 62.  
 ALLARD. Tonus musculaire et Tétanos électrique. *Soc. française d'électrothérapie*, Oct. 1903, p. 234.

# Review of Neurology and Psychiatry

---

## Original Articles

### THE STATE OF THE EYE MUSCLES IN ORGANIC HEMIPLEGIA.<sup>1</sup>

By S. A. KINNIER WILSON, M.A., M.B., B.Sc.

THE clinical observation that in many hemiplegias certain muscles on the paralysed side are apparently not affected, was long ago explained by Broadbent on the hypothesis that there is a bilateral representation in the cortex for those muscles on both sides of the body which usually act in concert, and that the degree of bilateral representation corresponds to the degree of bilateral use. It was accordingly accepted as a universal clinical fact, and the statement has been current in many text-books, that in organic hemiplegia those muscles which act together with their homologues on the other side are not touched. It may be questioned, however, whether the generalisation has not been accepted too literally, and whether the first enunciation of Broadbent's law did not include the possibility of all the muscles on one side of the body being affected, at least immediately after the onset of the hemiplegia. According to Gowers (1), in the muscles of bilateral use there is sometimes slight weakness for a short time after the onset, but the hemisphere of the same side is soon able to innervate them in full degree. He admits that the masseter on the paralysed side may not contract so strongly as the other, and that if the extraordinary muscles of respiration are brought into play there

<sup>1</sup> Résumé presented to the Neurological Society of Paris, Jan. 8th, 1904.

may be a distinct defect of expansion of the corresponding half of the thorax. More recent observation has further demonstrated the possibility of unilateral involvement of the abdominal muscles as well as modifications in the platysma on one side—though it may be questioned whether the latter ought to come under the category of bilaterally functioning muscles.

As far as the face is concerned, while the classic opinion has been that the lower facial group is practically always paralysed, the upper facial practically never, much clinical evidence is forthcoming to show that this difference is more apparent than real. Without mentioning earlier observations, one may refer to the work of Revilliod (2), who, relying on the orbicularis sign—the inability of the patient to close the eye on the paralysed side singly—demonstrated the not uncommon paresis of the upper facial muscles. The objection has frequently been made that this sign is of comparatively little value as many normal individuals are unable to close the eyes singly, at least with equal power and promptness. Revilliod's pupil Boiadjew (3) examined 750 normal individuals to make this point clear, and found that 64 per cent. could close each eye separately; whereas out of 25 hemiplegics examined by Pugliese and Milla (4), 22—i.e. 96 per cent.—were quite unable to do so. We must therefore conclude that Revilliod's sign is of considerable value in the diagnosis of an orbicularis paresis, especially as it has been shown to exist for some time after other facial symptoms have disappeared. The whole question has been discussed in detail by Mirallié (5), who is of opinion that in *every* case of paralysis (of cerebral origin) of the lower facial muscles, the upper facial are also affected, though usually to a much less marked degree.

The recognition and appreciation of these facts find a place in the most recent text-books. For instance, Von Monakow (6) says that careful examination of the paralysed side will reveal a delay in the closing of the eye and the wrinkling of the forehead, and that in fact *every* upper facial muscle has suffered a certain slight and transitory loss of power; while Oppenheim (7) agrees that the eye on the affected side may not be shut separately, or so tightly, or for so long as the other, that the eyebrow may be a little lower, and that the frontalis may not be contracted so powerfully. And while it is of course universally admitted that there remains always, or practically always, a very distinct

difference in the degree and duration of paralysis in the two groups, it will be seen that our conception of the nature of the muscular involvement in organic hemiplegia has altered. A restatement of our ideas is given by Déjérine (8) in the somewhat enigmatical expression: "the muscles are paralysed proportionately to their normal force." For Marie (9), too, Broadbent's law is insufficient. His argument is, that from the point of view of bilateral use, there can be very little difference between the orbicularis of the eyes and the orbicularis of the mouth, and yet they are far from being affected in the same way in a hemiplegia. For him, voluntary innervation (whether this correspond with innervation *via* the pyramidal paths or not) is much less highly developed for the upper facial than for the lower facial group; accordingly, if a cerebral lesion destroys the fibres in this "voluntary bundle," the degree of paralysis will be more marked in those muscles which have an abundant voluntary innervation than in those of which it may be said that their voluntary innervation is rudimentary.

But the field covered by this question extends still further, and in an interesting direction. A few cases have been published in which there was a unilateral ptosis undoubtedly of cerebral origin. In not all of the observations, however, was the ptosis the accompaniment of a hemiplegia. Reference may be made to the observations of Grasset (10) and Landouzy (11), of Surmont (12), Lemoine (13), Sigaud (14), Brissaud (15), Herter (16), Giannelli (17), Hartley Bunting (18), and Mirallié (19). It may be stated, then, on the authority of a few well authenticated cases, that an organic hemiplegia may, though exceedingly rarely, be accompanied by a certain amount of ptosis on the same side as the paralysis. It is argued by Brissaud that this apparent ptosis, if it occurs, is really due to involvement of the orbicularia. (In several of the recorded cases, for instance Giannelli's, it is definitely stated that the superior facial muscles were *not* involved.) Brissaud admits, to begin with, that the narrowing of the palpebral fissure on the affected side may be too pronounced to allow of its explanation as being merely a sign of general enfeeblement of the orbicularia. There is undoubtedly insufficiency of the levator palpebræ superioris; nevertheless, this muscle is innervated by the third cranial nerve, "whose function is never disturbed in a cortical lesion." As in a

musculo-spiral paralysis the flexors innervated by the median contract with less force, so in paralysis of the orbicularis, the levator palpebræ superioris suffers, the want of tone in the former depriving the latter of that support it must have in order to maintain the normal size of the palpebral fissure. Thus according to Brissaud, ptosis, when it occurs as a complication of hemiplegia, is explicable as being due in reality to the facial involvement.

This view has been strongly combated by Mirallié. Holding that the analogy between a musculo-spiral and a facial paralysis is inexact, he gives the details of 30 cases of hemiplegia, in no fewer than 8 of whom (*i.e.* in 26 per cent.) ptosis, or rather narrowing of the palpebral fissure, was present on the affected side. He is of opinion that the condition is much more frequent than is usually supposed, that it disappears quickly, and that it can only be explained on the theory that oculo-motor fibres are involved in the lesion, somewhere above the nuclei. In this paper, alluding to the third pair of cranial nerves, Mirallié says further that as of all the muscles supplied by them there is only one (the levator palpebræ superioris) whose movements are not absolutely and always bilateral, so there is nothing extraordinary in the fact that the eye movements preserve their integrity completely. The eye muscles function simultaneously, not singly. Nevertheless this author in conjunction with Desclaux (20), and the latter (21) also, still more recently, have now attempted to show, not merely that ptosis in organic hemiplegia is certainly due to involvement of the oculo-motor, but that in *every* case of this disease (if the face be touched at all), *all* the small muscles of the eye on the paralysed side are affected to a greater or less degree. In his thesis Desclaux has given the details of 18 cases of adult organic hemiplegia, recent or advanced. Employing for his purpose a little apparatus which will be described immediately, he first sought to establish the fact that in normal individuals there is never any difference in the power of homologous muscles of the two eyes. In his cases of hemiplegia he found, on the other hand, that the absolute power of each of the ocular muscles is diminished on both sides, but above all on the affected side, hence the muscles of the non-affected eye are stronger than their homologues; that this difference is in inverse ratio to the duration of the hemiplegia, and that the paralysis appears



to be directly proportional to that of the upper facial muscles.

In short, if we are to believe these latest results, we should expect to find in an ordinary hemiplegia—if the face is touched at all—not merely a paresis or paralysis of the musculature of the mouth and lips, but also, in every case, of the frontalis and orbicularis palpebrarum, of the levator palpebræ superioris, and finally of all the external muscles of the eye. And this, be it observed, not merely during the first few days or weeks after the onset: for several of the observations of the above authors relate to hemiplegics who had been paralysed for quite a number of years.

It is obvious that the question is of some clinical and anatomical interest. It would seem that we shall have to remodel our conceptions of cerebral paths and cerebral laws. Ptosis is a complication very far from common in organic hemiplegia, and its mode of production has not yet been satisfactorily elucidated. Theories differing widely enough have been advanced to account for it. And in addition the statement is now made that all the eye muscles supplied by the oculo-motor nerves are similarly involved, although there is apparently no connection between their paresis and that of the levator palpebræ superioris, but rather that of the superior facial group. This seeming inconsistency demands an explanation; but apart from that, when one has to deal with problems implicating the complex physiology of the ocular musculature, one must needs proceed warily. It is not so much the amassing of facts as the interpretation of them that is attended with difficulty, calling for the exercise of patience and judgment.

With a view to the accumulation of further clinical observations relating to the hypothesis of Mirallié and Desclaux, ten adults apparently normal, and ten hemiplegics were systematically examined. An apparatus much the same as that described by these authors was used.

It consists essentially of a tube of zinc 24 centimetres long, 4 centimetres in diameter at the end which is to be applied to the eye; the other has a diameter of one centimetre. It is mounted on a little stand of a convenient height, so that when the patient is seated at a table and leans forward, the tube is about on the same level as his eyes. Now in ordinary binocular

vision, when an individual regards an object at a little distance, the eyes are so directed that the rays coming from that object fall on that part of each retina which is the most sensitive: this is of course the fovea centralis, and if the eyes are in perfect equilibrium, perfect vision results. Should the rays not strike points in the two retinæ which correspond absolutely, then imperfect vision or diplopia, in greater or less degree, results. The eyes, however, may not be in perfect equilibrium, yet diplopia may not ensue. If, for instance, the musculature of one eye is for any reason defective, the rays coming from an object may not fall on absolutely identical spots in the two retinæ. Diplopia would naturally result, but the sound eye unconsciously receives an additional innervation sufficient to turn the globe—perhaps in an infinitesimal degree—so that it does receive the rays on a spot corresponding to that in the other (impaired) eye. Thus the diplopia is overcome: the deviation is superable: but the equilibrium is not perfect, it is forced. We may again deliberately produce diplopia by placing prisms before the eye so that the rays are deviated in any given direction. An attempt may be made to overcome this diplopia by dint of forcing the muscles of the other eye to bring the globe into a similar position, or it may be insuperable.

Use was made of these facts in the present examination. The conical tube was placed before one eye, quite close to it, so that the patient could not possibly do else than regard any given object through the cone. At a distance of about 5 metres was a small white disc,  $4\frac{1}{2}$  centimetres in diameter, on a dark background, and this was looked at steadily through the tube. Of course there was ordinary binocular vision, when both eyes were open, and the globes being in equilibrium there was no diplopia. But the field of vision for one eye was fixed, and in the experiments that followed, the patient was unable to move that eye from its position, i.e. as long as he continued to have the cone applied close to it, and to regard the white disc along the axis of the cone. In front of the other eye were now placed one after the other a series of prisms, graduated in degrees from one to twelve. These prisms were circular in form, so as to fit into the ordinary pair of spectacles one employs to hold lenses when testing for myopia, etc. Beginning with the feeblest, No. 1, it was placed in front of the eye with the base directed outwards.

The rays coming from the disc did not now strike the retinae in corresponding points: they were deviated by the prism towards its base, and so they impinged on a retinal area situated a little further out than that on which were falling the rays reaching the other eye. To avoid the diplopia which would naturally ensue, the patient's eye muscles contracted so as to bring it into a position whereby the rays again fell on a spot identical to that in the other eye, viz., on the fovea. The internal rectus is so attached that it rotates the anterior portion of the globe inwards and the posterior outwards, about a vertical axis. It was, therefore, the internal rectus which, by rotating the retina outwards, enabled it to catch the deviated rays again on the fovea centralis. Thus by means of increased innervation of this muscle, the diplopia was overcome. In other words, we have a test of its power. One then proceeded to place the next prism in front of the eye, and so on, until at last a diplopia was produced which the patient, in spite of every effort, was unable to overcome: it was permanent. Again assuring oneself that with the preceding prism the patient could approximate the two images, one had an index, in the number of this prism, of the power of the internal rectus on that side. Similarly by turning the base of the prism to the inside, one obtained the value of the external rectus in degrees of the graduated prisms. By turning the base upwards and downwards respectively, figures for the elevators and depressors of the eyeball were likewise procured. It remained to transfer the cone to this eye, and to proceed in an absolutely identical manner to examine the other. Finally, a set of figures for the two eyes was got, whereby one might compare the relative value of homologous muscles of the eyes evaluated in degrees.

Though this method for testing the power of the eye muscles may appear simple and convenient, it is associated with certain difficulties, both theoretical and practical, which must be mentioned. To begin with the latter: apart altogether from obvious drawbacks inherent in any technical examination, such as the inability of the patient to appreciate what is demanded of him, his answering at random, his failure to regard steadily the object as seen through the instrument, his evident misinterpretation of what he sees, it must be remembered that this test demands a conscious effort, a deliberate attempt by straining the eyes to

make the diplopia disappear. This naturally enough produces fatigue, and some subjects are more easily fatigued than others. It was found on several occasions that if one commenced by testing all the muscles of one eye first, the muscles of the other eye in their turn responded more feebly. Accordingly it became necessary either to wait a little or to repeat the examination in an inverse order on another occasion, in order to be sure of correct results. Sometimes after straining to overcome one of the prisms, on its removal the patient continued to see double, indicative of a mild paresis of an individual muscle or of several muscles, and again one had to discontinue the examination.

A more serious difficulty in this method of procedure is the demand it makes upon what one might call the innervating power of the individual, to be exerted, be it observed, along paths probably never before similarly exercised. In other words, muscles which always act simultaneously and in concert, are by this procedure dissociated in their action, and the individual is called on to energise certain of them singly in a way he has not been accustomed to. A subjective element is therefore introduced which it is difficult to eliminate, and the personal equation becomes an important factor. So little may the subject be able to produce the desired effect, so inaccurately may he apply his will-power in an unwonted direction, that, as I have satisfied myself was the case in several instances, in the attempt to make the double images produced by the prism converge, he may in reality drive them still further apart.

Theoretically, it must be remembered that this method of examination is essentially a test for convergence and divergence, *not* for adduction and abduction, and so on. The difference between the two has been very justly pointed out by Fuchs (22). When side to side movements are to be made, the eye can be abducted until the external margin of the cornea almost touches the external angle of the eye, while the outward movement of the eye in performing divergence is but a minimal one. Evidently then, as far as actual movement is concerned, we are employing a test which does not allow of much scope in this direction; if we are to judge of the paralysis of any given muscle by the limitation of its range of movement, we are not using the best means for this end. Moreover, adduction and convergence follow quite different laws. Hence it became necessary to corroborate and

compare results by the use of a test for lateral and vertical excursion of the eyeball. The perimeter as a means of determining the field of fixation (not the field of vision) was resorted to. Reference will be made to this later. To satisfy oneself as to the actual value of the prism method, then, ten apparently normal individuals were examined, all of whom happened to be members of the medical profession. It is therefore a superfluous remark that as far as the appreciation of the technique was concerned there could be no difficulty. Nevertheless the results obtained demand investigation by reason of their variety.

*Observation 1. Age 26.*

	Right.	Left.
Convergence	12°	12°
Divergence	4°	4°
Upward deviation	2°	2°
Downward deviation	2°	2°

*Observation 2. Age 30.*

Convergence	6°	6°
Divergence	5°	6°
Upward deviation	6°	6°
Downward deviation	5°	5°

*Observation 3. Age 31.*

Convergence	12°	12°
Divergence	4°	4°
Upward deviation	5°	5°
Downward deviation	4°	5°

*Observation 4. Age 24.*

Convergence	5°	5°
Divergence	2°	2°
Upward deviation	1°	1°
Downward deviation	2°	2°

*Observation 5. Age 34.*

Convergence	12°	12°
Divergence	4°	4°
Upward deviation	1°	1°
Downward deviation	1°	1°

*Observation 6. Age 28.*

	Right.	Left.
Convergence	7°	6°
Divergence	7°	6°
Upward deviation	2°	3°
Downward deviation	2°	3°

*Observation 7. Age 26.*

Convergence	3°	3°
Divergence	3°	3°
Upward deviation	2°	2°
Downward deviation	2°	2°

*Observation 8. Age 25.*

Convergence	10°	12°
Divergence	3°	3°
Upward deviation	1°	1°
Downward deviation	2°	2°

*Observation 9. Age 25.*

Convergence	7°	7°
Divergence	5°	5°
Upward deviation	1°	1°
Downward deviation	1°	1°

*Observation 10. Age 27.*

Convergence	12°	12°
Divergence	6°	6°
Upward deviation	4°	4°
Downward deviation	5°	5°

It will be seen at once as a general rule that strong prisms can be overcome by convergence, weaker ones by divergence; while usually only feeble prisms can be overcome by vertical deviation of the eyes.

It will be seen further, that while some subjects examined could by convergence overcome a prism of 12° with either eye, others failed to overcome a very much feebler one (3° or 5°). Are we to interpret this as signifying that there actually exists this difference between two individuals in the "power" of those muscles which produce convergence, although to all ordinary tests there is no impairment of vision in either? It is more likely that one subject was simply more expert than the other. And finally,

it is evident that there may exist in the same person slight differences, as tested by this method, between homologous muscles of the two eyes. Thus not merely must the categorical statement of Desclaux be modified, but the value of his comparison with the results obtained in the case of hemiplegics is lessened.

As far as the latter are concerned, the following order of examination was adhered to: First, any obvious facial asymmetry or paralysis was remarked, and the facial muscles were systematically tested by ordinary methods. In investigating the upper facial group, special attention was paid to the points raised by Mirallié: is the position of the two eyebrows the same? does the outer extremity of one droop more than that of the other? can each eye be closed separately? are the upper facial movements on one side as extensive as those on the other? or as quick? or as strong? is there any difference in tonicity? or in movements against resistance? Next, the eyes themselves were examined for any obvious morbid appearance, *e.g.* mydriasis. Ocular paralyses of a more pronounced type were tested for, and ordinary ocular movements elicited. The last step was to satisfy oneself (by test types, etc.) that no diplopia existed, or to correct any simple defect of vision before proceeding to employ the prisms.

*Observation 11.* Male, aged 45. Right hemiplegia with some troubles of speech. Duration 3 years. No ptosis. Can close eyes separately. No asymmetry upper part of face.

	Right.	Left.
Convergence	5°	5°
Divergence	2°	2°
Upward deviation	1°	1°
Downward deviation	1°	1°

*Observation 12.* Male, aged 40. Left hemiplegia of 13 years' duration. Slight facial asymmetry involving upper part of face. No ptosis. Can close eyes separately.

	Right.	Left.
Convergence	3°	2°
Divergence	3°	2°
Upward deviation	1°	1°
Downward deviation	1°	1°

*Observation 13.* Male, aged 64. Left hemiplegia of 3 years' duration. No appearance of upper facial group being involved.

	Right.	Left.
Convergence	4°	4°
Divergence	2°	2°
Upward deviation	1°	0°
Downward deviation	1°	0°

*Observation 14.* Male, aged 76. Right hemiplegia for 6 years. No appearance of upper facial group being involved. Slight asymmetry of mouth.

	Right.	Left.
Convergence	2°	2°
Divergence	2°	2°
Upward deviation	1°	1°
Downward deviation	1°	1°

*Observation 15.* Male, aged 41. Left hemiplegia: duration 4 months. Cannot close eyes separately. Nothing else to note in upper part of face.

	Right.	Left.
Convergence	4°	3°
Divergence	4°	3°
Upward deviation	3°	2°
Downward deviation	3°	2°

*Observation 16.* Male, aged 68. Left hemiplegia: duration 6 days. Upper facial group slightly impaired. No ptosis. Cannot close eyes separately. Left pupil larger than right.

	Right.	Left.
Convergence	4°	4°
Divergence	2°	2°
Upward deviation	0°	0°
Downward deviation	1°	0°

*Observation 17.* Male, aged 70. Left hemiplegia: duration 8 years. Cannot close eyes separately. Slight relative narrowing left palpebral fissure.



	Right.	Left.
Convergence	3°	3°
Divergence	1°	0°
Upward deviation	0°	0°
Downward deviation	0°	0°

*Observation 18.* Male, aged 55. Right hemiplegia: duration 2 years. No ptosis. Can close eyes separately. No sign of upper facial weakness.

	Right.	Left.
Convergence	6°	6°
Divergence	4°	4°
Upward deviation	2°	2°
Downward deviation	2°	2°

*Observation 19.* Male, aged 66. Left hemiplegia: duration 2 years. Slight upper facial asymmetry: left eyebrow a little lower than the right. Can, however, close eyes separately.

	Right.	Left.
Convergence	3°	2°
Divergence	3°	2°
Upward deviation	1°	1°
Downward deviation	1°	1°

*Observation 20.* Male, aged 45. Left hemiplegia: duration 4 years. Cannot close eyes separately. Nothing else to note in upper part of face.

	Right.	Left.
Convergence	4°	4°
Divergence	4°	4°
Upward deviation	1°	1°
Downward deviation	2°	2°

When one comes to consider these results analytically, it will be seen that they entirely fail to confirm the theory of Desclaux. Cases of hemiplegia of very varying length of duration were chosen, occurring at varying ages, yet in no case was a difference between the ocular muscles of the two sides at all marked. It is true that six times out of ten there was a difference, but it never amounted to more than one degree, and in the case of apparently normal individuals a similar difference betrayed itself in several instances. It is to be remarked, no

doubt, that the side of diminished force, such as it is, in the cases of hemiplegia, was always the paralysed one, but it is impossible to base a theory on a foundation so slender. One of the observations (No. 16) seemed to furnish a test case, the lesion being so recent, and the upper facial muscles being certainly, though slightly touched, nevertheless it failed to confirm Desclaux' hypothesis. Some stress has been laid on the fact that as a whole the power of the ocular muscles is diminished on both sides (although more on the affected side), and the observations given above make no exception. One does not find the No. 12 prism overcome, nor is this figure even approached, the highest one overcome by convergence being the one of  $6^{\circ}$ . But the presumption that this apparent diminution or weakness is essentially the result of the lesion, is rendered somewhat dubious by the fact that the condition before the hemiplegia is of course unknown; further, some of the patients were old and naturally enfeebled, and incapable of efforts demanding concentration; nor must it be forgotten that one or two of the normal cases examined showed a similar inability to overcome the prisms of higher degree. Again, there is an approximation, to a certain extent, of the figures for convergence and divergence respectively, which has also been remarked by Desclaux. But, again, this is no more than was observed in one or two individuals apparently normal.

The fact has been already mentioned that the prism method tests the ability of the eye to converge, diverge, etc., not to adduct or abduct. The terms are by no means synonymous, divergence, for instance, being a more complicated proceeding, involving a more complicated muscular action than abduction. It is distinctly unfortunate that in the thesis of Desclaux no apparent notice is taken of this fact; instead of convergence, etc., mention is always made of the individual *muscles*, the internal, external, superior and inferior recti, as if they were the only muscles involved in the production of the phenomena. No reference whatever is made to the superior and inferior oblique muscles, though they play an integral part in all ocular movements with the exception of adduction. The figures given are supposed to represent the actual power of individual eye muscles expressed in degrees, instead of representing, as they do, the sum of a complex action.

As a test for individual eye muscles, the perimeter was employed to measure the excursions of the eyeball. Two letters on a small white card were moved in from the periphery towards the centre until the patient (one eye being closed) was able to distinguish and read them with the other. It was essential to have his head steady and in the middle of the perimetric arc. Thus by altering the position of the arms of the instrument, one could map out the field of fixation. Not all of the hemiplegic cases were examined, but no one of those (six) who were, showed any marked deviation from the normal.

The conclusion one comes to is that it is impossible to argue for an involvement of the ocular muscles in an organic hemiplegia on the ground of slight differences with the prism method. It has been shown that these can not be eliminated because of the subjective element inherent in the examination. To bear this out, it will be sufficient to cite the results in three cases of complete hysterical hemiplegia which were carefully examined by both methods.

*Observation 21.* Male, aged 59. Right hysterical hemiplegia of 3 days' duration. Cannot close eyes separately. No diplopia, but dimness of vision (corrected). No appreciable facial asymmetry. Tongue deviated to the right.

	Right.	Left.
Convergence	0°	6°
Divergence	0°	4°
Upward deviation	0°	0°
Downward deviation	0°	1°

*Observation 22.* Male, aged 54. Right hysterical hemiplegia: duration 16 days. Cannot close eyes separately. Marked dimness of vision (corrected). Tongue deviated to the right. No obvious facial asymmetry.

	Right.	Left.
Convergence	0°	3°
Divergence	0°	3°
Upward deviation	0°	1°
Downward deviation	0°	1°

*Observation 23.* Male, aged 74. Left hysterical hemiplegia: duration 30 years. Can close eyes separately. Nothing

in face. Tongue most markedly deviated to the left. Has dimness of vision. No diplopia.

	Right.	Left.
Convergence	2°	1°
Divergence	2°	1°
Upward deviation	2°	0°
Downward deviation	2°	0°

These cases of hysterical hemiplegia show a marked difference between the figures for the sound and for the paralysed side. In the first two cases the patient was quite unable to overcome even the feeblest prism; diplopia was at once produced, and it was permanent as long as the prism was held in front of the eye. There seemed to be in each of the cases a complete inability to make the conscious effort required to converge, with the result that the difference between the two eyes was much more marked than in any of the organic cases. On the sound side there was no marked deviation from the ordinary condition. Testing of the eye muscles for bilateral movements, and unilaterally with the perimeter, revealed nothing of moment, except that in the latter case undoubtedly the excursions of the eyeball were somewhat more limited than in any of the organic cases, and one may justly, I think, find in this a corroboration of the results from the prism method.

In any case, support is given to the view that unilateral convergence or divergence demands in its performance a subjective element which is bound to vary greatly within certain limits, and the failure of the hysteric to mount the feeblest prism reveals his inability to innervate.

It is, however, or it may be somewhat precarious to argue from hysterical to organic cases. In the face of our ignorance of the seat of the lesion in hysterical cases, and in view of the most bizarre and apparently inconsistent phenomena which they may reveal, it is for the present undesirable to lay too much stress on the observations in the functional cases. Although every care was taken to eliminate error, it is conceivable that they do not represent the actual state of affairs. In an article by Babinski (23) on the differential diagnosis between functional and organic hemiplegia, it is stated that there is never any effacement of wrinkles or droop of the eyebrow, nor is there any impairment in the ordinary bilateral movements of whistling, speaking, etc.

In a series (24) of eight cases of functional pareses or paralysis in the facial and hypoglossal groups occurring in hysterical hemiplegia, no particular mention is made of involvement of the upper facial muscles; the lower, on the other hand, being very considerably impaired. It might therefore be expected that there would be no involvement of the eye muscles, which always act in concert. As far as bilateral movements are concerned, there certainly was no impairment, but a pronounced unilateral weakness was revealed which was not paralleled by the condition of the levator palpebræ superioris or upper facial muscles.

Be this as it may, whether it be—as is most likely—merely an exaggeration of the real difficulty in Desclaux' method or not, it has been seen that little support is given to the view that in all organic hemiplegias where the face is touched at all, the muscles supplied by the third, fourth and sixth pair on that side are also involved. It has been seen, further, that there is no apparent relationship between the condition of the superior facial and of the eye muscles. While in more than half of the cases examined (and eight additional observations are not recorded because of some uncertainty as to the accuracy of the results obtained) there was evidence—if we are to accept Revilliod's sign and assume in health a *rigorous* facial symmetry—that the upper facial muscles had been paresed, there was no corresponding paresis of the oculo-motor muscles.

These facts find their easiest interpretation in Broadbent's law, however one may choose to express it. To use an old classification of Hughlings Jackson, the muscles of the body may be divided into those which must act bilaterally, those which may act alternately, and those which can act independently. All are represented in each side of the brain, the first group, however, being equally represented, the second less equally, and the third very unequally. But the whole difficulty consists in determining what are the movements which are essentially bilateral, that is to say, under the control of cortical areas of true bilateral representation; what are the movements bilaterality of which is doubtful or incomplete; what are those which do lack this character, although perhaps still considered in the category.

The satisfactory consideration and elucidation of this question demand all the anatomical and clinical evidence possible, together with all the experimental results of the physiologist.

Confining ourselves solely to those muscular groups which for the moment interest us, the fact remains that in ordinary adult organic hemiplegias there is practically always a marked difference in the degree of paralysis of the superior and inferior facial muscles. Anatomically, the medullary and cerebral paths of the fibres supplying these two divisions cannot be very different. Their nuclear origin is the same [van Gehuchten (25), Marinesco (26)], and above the pons, while Charcot and Pitres (27) consider that all cortical allocation of a motor centre for the upper facial muscles is premature, later opinion tends towards its localisation in the lower part of the ascending frontal convolution, above the centre for the mouth and tongue, in the neighbourhood of the posterior extremity of the second frontal (28). The explanation of the variation in degree of paralysis must therefore be largely physiological. The bilateral representation of the orbicularis palpebrarum, frontalis, etc., must be more advanced. To use Marie's phrase, their voluntary innervation must be more rudimentary.

The same is true of the levator palpebræ superioris. Here again evidence is forthcoming to find a cortical centre for this muscle in the angular gyrus, but it is a much more dubious localisation (29). When one considers how common hemiplegia is, and how exceedingly rare ptosis, as a concomitant phenomenon of cerebral origin, one is struck by the disproportion between the two conditions. It cannot be argued that the latter symptom is one which, because it is early and transient, is likely to escape observation. The vast majority of patients suffering from hemiplegia are seen within a very short time after the onset, and even a slight degree of ptosis, if it had been at all frequent, as frequent as some suppose it is, would have been known as a classic sign long ago. The conclusion is that for this muscle voluntary innervation is still more rudimentary. Wernicke (30) holds that the excessive rarity of a true unilateral ptosis in organic hemiplegia renders it very improbable that the levator has a unilateral innervation from the cortex. Further, for this muscle and for the upper facial, there are almost certain to be individual variations.

Ever since the first experiments of Ferrier the attention of physiologists has been almost continuously directed to the question of the cortical centre or centres for movements of the

eyes. And the general consensus of opinion is that the ocular muscles are absolute in their bilateral representation and innervation.<sup>1</sup> Except under special experimental conditions beyond the present subject, no cortical stimulation produces unilateral movements of the eyes. It is impossible to individualise the oculo-motor muscles; no one of them is in itself and by itself under the voluntary control of the cortex. Single or complete unilateral ocular palsies—of other than nuclear or peripheral origin—have never been described.<sup>2</sup> As far as the tract of cerebral fibres is concerned, we are still very much in ignorance. Ocular movements can be stimulated from widely separated areas of the cortex. According to some, those fibres which pass from the occipital lobe forward to the eye nuclei do not belong to the pyramidal system. Cases have been described quite recently in which following gross unilateral lesions of the cortex, there was marked degeneration of the fibres reaching the seventh nucleus from above, yet clinically there was no corresponding paralysis of the facial muscles (31). It must be evident, *a priori*, that we should not expect a necessary involvement of all of these oculo-motor fibres in every case of organic hemiplegia involving the extremities and the face. On anatomical and physiological grounds, therefore, unilateral paralysis of the eye muscles of cerebral origin is, to say the least, most improbable; and though in many historical instances clinical evidence has led the way to new discoveries, in this case undeniable evidence is still wanting.

It is my pleasant duty to thank Professor Pierre Marie, not merely for the material afforded me at Bicêtre for carrying out this investigation, but also for his constant and kindly interest and advice.

#### CONCLUSIONS.

1. The attempt to demonstrate unilateral ocular paralysis or paresis in adult organic hemiplegia with the prism method is unsatisfactory.

<sup>1</sup> For a discussion of the whole question, cf. Soury, "Le système nerveux central," 1899, vol. ii., pp. 926-968 and 1406-1420.

<sup>2</sup> Gowers (*British Med. Journ.*, May 1902) has described several cases of myasthenia associated with aberrant unilateral or bilateral ophthalmoplegia. This may be idiopathic in the muscles, or it may be "central." By "central" is probably meant nuclear.

2. In normal individuals, as tested with this method, there may exist differences in the power of homologous eye muscles.

3. The difference in cases of hemiplegia is not necessarily any greater.

4. The argument that any difference in hemiplegia cases is due to the lesion is unjustifiable.

5. In hysterical hemiplegia the difference in power of convergence, etc., may be marked.

6. There is no necessary relation between the condition of the eye muscles and the degree of paralysis of the upper facial group.

7. For anatomical and physiological reasons, further, the question of the possibility of unilateral ocular palsies of cerebral origin must be considered with reserve.

#### REFERENCES.

1. Gowers. "Diseases of the Brain," 1887, p. 55.
2. Revilliod. *Rev. méd. de la Suisse Romande*, 1889.
3. Boiadjew. *Thèse de Genève*, 1892.
4. Pugliese and Milla. *Riv. sperim. di freniat.*, 1896, p. 805.
5. Mirallié. *Archives de Neur.*, 1899, p. 1 et seq.
6. Von Monakow. "Gehirnpathologie," p. 288.
7. Oppenheim. "Lehrb. d. Nervenkrankh.," 3rd edit., p. 608.
8. Déjérine, in Bouchard's "Path. générale," 1901, vol. v., p. 483.
9. Marie, in Brouardel and Gilbert's "Traité de Médecine," 1901, vol. viii., p. 460.
10. Grasset. *Progrès Médical*, 1876.
11. Landouzy. *Arch. gén. de. Méd.*, 1877.
12. Surmont. *Thèse de Lille*, 1886.
13. Lemoine. *Rev. de Méd.*, July 1887.
14. Sigaud. *Progrès Médical*, 1887.
15. Brissaud. *Progrès Médical*, Dec. 1893.
16. Herter. *Journ. Nerv. and Ment. Dis.*, Jan. 1895.
17. Giannelli. *Riv. di Psicol. Psich. e Neur.*, 1897.
18. Hartley Bunting. *Lancet*, Aug. 1898.
19. Mirallié, *loc. cit.*
20. Mirallié and Desclaux. *Revue Neurologique*, 1903, p. 649.
21. Desclaux. *Thèse de Paris*, 1903.
22. Fuchs. "Ophthalmology," Eng. edit., 1900, p. 592.
23. Babinski. *Gaz. des Hôpitaux*, May 1900.
24. Koenig. *Neur. Centralb.*, 1892, p. 337.
25. Van Gehuchten. "Anat. syst. nerv. de l'homme," 3rd edit., vol. ii., p. 109.
26. Marinesco. *Rev. Neurologique*, 1898.
27. Charcot and Pitres. "Centres moteurs corticaux," 1895, p. 151.



28. Cf. v. Bechterew. "Die Leitungsb. im Gehirn u. Rück." (French edit.), 1900, p. 664.

29. Cf. Lemoine, *loc. cit.*

30. Wernicke. "Lehrbuch d. Gehirnkrankh.," vol. i., p. 323.

31. Sand. *Arbeit aus d. neur. Instit.* (Wien. Univ.), 1903, H. 10, p. 208.

## VARIATION IN ITS RELATION TO THE ORIGIN OF INSANITY AND THE ALLIED NEUROSES, BEING THE MORRISON LECTURES FOR 1904.<sup>1</sup>

By JOHN MACPHERSON, M.D., F.R.C.P.E., Commissioner in Lunacy  
for Scotland.

### LECTURE I.

#### [AUTHOR'S ABSTRACT.]

IF a number of individuals of any species be compared with one another, it will be found that they all show differences from one another in size, shape and colour; and if they be animals, in their mental characteristics. Offspring differ from their parents, brothers from brothers and sisters, and brothers from one another. These differences constitute what is known as "Variation," and it is upon variation that the evolution of organic life—apart from its primary cause—essentially depends. It was pointed out that the use of the term "Heredity," as implying that parents hand down to their offspring characters which essentially belong to themselves is false and misleading, and is based upon the analogy of the descent of property in families. The only tenable position is that descent is not through the parents' body at all, but through the germ cells. Variation primarily arises as a result of sexual reproduction, and although that is disputed by many eminent authorities, the fact remains that we do not know variation except as a direct or indirect accompaniment of sexual reproduction. With regard to the influence of the environment, it was held that in no case can external conditions call out properties with which the individual was not endowed at the act of conjugation. The extent of variation in wild nature was dealt with at considerable length, and it

<sup>1</sup> Delivered before the Royal College of Physicians of Edinburgh on the 25th, 27th and 29th Jan. 1904.

was shown by means of diagrams and quotations that the prevalent opinion that variation is confined to domestic plants and animals is erroneous. On the other hand, certain species tend to vary abnormally more than others. Thus, frequency of extra teeth are certainly more common in domestic cats and dogs than among their wild congeners, but they are not more so than among the higher apes and seals. Again, digital variations are common in the horse, cat and pig, rare in the dog and sheep, and unknown in the ass. The measurement of variation was insisted upon as the only means of securing uniformity of observation. If, out of a large number of observations, it is asserted that five per cent. vary plus or minus from the average of the whole, there is thus afforded a numerical expression of the degree of variation which can readily be compared with other expressions of a similar kind. Illustrations of this form of measurement were given, and the following table from the Report of the Anthropological Committee of the British Association, for 1883, of the stature of 6194 English adult males, was taken as an example:—

STATURE OF 6194 MALES.

Height in Inches.	57	58	59	60	61	62	63	64	65	66	67	68	69	70	71	72	73	74	75	76	77
Frequency	1	3	12	39	70	123	320	524	740	881	918	886	753	473	254	117	43	16	9	1	1

From the above table it will be seen that the greatest of stature for English males is 67 inches. The most common, that is, the most normal individuals, group themselves around this figure, which is the "mode," or mean. As we proceed further from the mean to right or left (plus or minus) the frequency becomes less and less, and the individuals become less typical of the race, or less normal. In dealing with such a comparatively small number of individuals, it does not necessarily follow that the individuals at each extremity of the table are abnormal; but if, instead of 6000, we were to take 6,000,000 persons, we should undoubtedly find at the one extremity pathological dwarfism and at the other pathological gigantism. Any other character, such as weight, hair colour, eye colour, or mental ability, could be grouped in a similar manner, and if thrown

into the form of a chart or curve, it could at once be determined whether the distribution was normal or in accordance with the calculus of probability. If the chart or polygon of frequency can be accurately fitted with a normal curve, the chances are greatly in favour of such a quality being genetic in origin. The paucity of the individuals at each extremity of the table is due to two causes or laws: (1) to the law of genetic selection, and (2) to the law of regression towards mediocrity. Professor Karl Pearson has formulated the law of genetic selection in the following terms: "Fertility is not equally distributed among all individuals, but for stable races there is a strong tendency for the character of maximum fertility to become one with the character which is the type." Mr Francis Galton has shown that on the average offspring are more mediocre in any selected character than their parents, and that whether the parents possess a character in small degree or in great, the offspring always tend to resemble the mean of the population. It is for these reasons that the most typical members of a race are the most numerous. It is the same with mental and moral characters. Professor Pearson<sup>1</sup> has recently shown that mental characters are inherited in precisely the same way as physical. That being so, they must be distributed in the population in a similar manner. By means of correlation tables and curves prepared by Professor Pearson, the distribution of mental ability among 2014 school-girls from ten to fourteen years of age was shown. The same method was applied to the distribution of mental ability among 107,000 Glasgow school children, of whom 235 were last year reported to be mentally defective. From the preceding and other facts, it was concluded that mental characters are distributed in the population just as is stature; that a minimum of any mental quality is possessed by a few individuals occupying one extremity of any grouped table or curve; a maximum by a few individuals at the opposite extreme; and that the most normal (the most numerous) individuals group themselves around the mode, or mean.

<sup>1</sup> Huxley Memorial Lecture, 1903.

## LECTURE II.

## MENTAL DEFECT.

Mental defect is a variation characterised by the absence of one or more of the numerous faculties or sub-faculties which we recognise in the normal human being, child or adult. Mental defect is wholly or in part correlated with the development of the physical organisation, especially with that of the nervous system, and it is rare to meet with imperfect congenital structure of the nervous system in the absence of other imperfections of the body. These imperfections of the body are technically known as physical stigmata. It is acknowledged that the more grave the incidence of the mental defect, the more numerous and the more pronounced are the physical malformations. There are also to be considered the mental stigmata, such as epilepsy, hysteria, alcoholism, chorea, and the various tics and obsessions which are the outward manifestations of the underlying defects in the nervous system. We are as yet far from any knowledge of the intimate correlation between structure and function. The most important attempt to correlate mental power with the structure of the cerebral cortex has been made by Dr J. C. Bolton (*Mott's Archives of Neur.*, 1903). He holds that the depth of the pyramidal layer of nerve cells in the prefrontal cortex varies inversely with the amount of amentia or dementia present in each case; that the degree of its development in normal infants and congenital aments varies directly with the mental power of the individual; and that the degree of its retrogression in demented patients varies directly with the amount of existing dementia.

Basing upon the correlation between physical stigmata and mental defect, the lecturer proceeded to pass in review the various forms of viable and non-viable monsters, and then proceeded to give a description of the graver physical anomalies which are found among idiots and imbeciles. Emphasis was laid upon the frequent occurrence of these anomalies among the lower animals, among primitive savages, and in early historical times. Reference was also made to their hereditary transmission, to the relative proportion of the sexes, especially in the case of monsters, and to the frequency of twinning in connection with the births of monsters and malformed individuals. The hereditary tendency

to the transmission of plural births in families was shown and also the morbidity of the process among the higher animals, including man. It was then demonstrated that plural births are common in families in which idiocy and imbecility is also common. An article by Sir Arthur Mitchell (*Med. Times and Gaz.*, 1862) was quoted, in which it was proved from 443 cases that a much larger proportion of idiots and imbeciles are twin born than of normal persons; that among the relatives of idiots and imbeciles twinning is very frequent; and that in families where twinning is common, bodily deformities of excess and defect likewise occur with frequency. Turning next to the relative proportion of the sexes in monsters, the subjects of anomalies, idiots, etc., it was shown on the authority of Van Lenhosseck and Beard, that while the normal proportion of the sexes at birth is roughly 106 males to 100 females, when account is taken of abortions down to the end of the second month, when sex can be distinguished, the proportion of males to females is nearly as 200 is to 100. Now, the great cause of abortion, apart from placental and maternal influences, is an abnormality in the embryo, and we must therefore conclude that but for the fact of abortion the relative number of human monsters would be much greater than it is. In the *Edin. Med. Journal* for 1866, Sir Arthur Mitchell clearly shows: (1) That the proportion of male to female idiots is as 100 is to 79. (2) That the excess of males is most marked in the graver forms of the disease. (3) That even among imbeciles it will probably be always found that males exceed females, though not to a great extent. We have now traced the cause of human anomaly and mental defect very near its lair, and we are pretty safe in assuming that, however arising, it depends primarily or secondarily upon abnormalities in the germ cells which the embryo receives into itself at an early stage in its growth, and we may assume that for some as yet unknown reason the male germ cells are more liable to abnormality than the female. There are only three things known with certainty regarding the cause of abnormalities: (1) They are capable of being experimentally produced. (2) They are innate in the germ plasma itself, otherwise they could not be regularly transmitted. (3) They tend to occur in certain species, families and stocks with almost numerical regularity. The experimental production of anomalies

is accidental, and is caused by an extraneous influence retarding the development of the embryo in the egg or uterus. It throws no light, therefore, upon the more subtle and more important power of the germ cell to convey the latent possibility of malformation. We may fancy in our own minds an explanation of the loss of fingers or the absence of a head by some theory of arrested development *in utero*, but we can scarcely argue from that to a similar explanation of an additional number of toes and fingers, or of two heads. We may account by some theory for the causation of idiots and monsters by toxins circulating in the mother's blood, but we cannot legitimately extend that theory so as to explain the persistent transmission of similar phenomena in families through the male line. Therefore, transmitted abnormalities must be innate in the germ plasm.

### LECTURE III.

#### THE NEUROSES.

As it was impossible to consider in one lecture all the neuroses, the subject was limited to remarks upon epilepsy, hysteria and alcoholism.

Epilepsy was defined as a variation characterised by convulsions or by sudden temporary impairment or loss of consciousness. It does not depend upon organic disease of the brain, or upon reflex irritation of the nerves, although these may occasionally start it. Certain factors in the pathogeny of the disease, especially the probable toxic basis, were discussed. Emphasis was laid upon its close association with mental defect, bodily malformation and insanity, and it was held to be a stigma of degeneration; an unmistakable sign of the neuropathic constitution. Its occurrence among various species of animals was discussed, and it was shown that among mankind it is universally distributed and has no racial or geographical limit. In Europe it was estimated from various sources that 1·5 per 1000 inhabitants are subject to it, and reasons were given for believing that the proportion of epileptics in Scotland was probably not less than that in Europe.

Hysteria was defined as a purely psychical variation charac-

terised by emotional excitement, sometimes by hallucinations of sight and hearing, by peculiar convulsive muscular movements, and by hypnotic states during which the subject acts or speaks impulsively either under the influence of auto- or inter-suggestion. The undoubted occurrence of hysterical excitement, of catalepsy, and even of hysterical paresis among the lower animals was discussed. The prevalence of hysteria among primitive savages was fully entered into, and it was shown that estimated by the frequent references to it in the writings of travellers, lay and medical, hysteria must be not only the most common of all the neuroses, but the most widely diffused over the world. Judging from the comparative frequency of these references, the opinion may be formed that the inhabitants of the Arctic latitudes of the eastern hemisphere are particularly subject to it. Brief accounts were given of "Latah" as it occurs among the Malays and of certain epidemic and endemic psychopathies in various parts of the world.

Alcoholism was defined as an abnormal craving for artificial mental states which can be produced by drugs, the most commonly used of which is alcohol. The subject was treated in connection with an endeavour to place alcoholism among the neuroses, and the following bonds of resemblance between alcoholism and the neuroses were given: (1) It is strongly hereditary. By that is meant that the peculiar mental constitution which craves after intenser states of consciousness is inherited. That this peculiar mental constitution is not produced by the drinking habits of parents was proved by many arguments, but especially by the fact that descent is through the germ cells alone. (2) The age at which the drinking habits appear was from numerous statistics shown to be from 17 to 25 years of age, which is also the age of incidence for the other neuroses. It was also shown that there is strong reason for believing that like the other neuroses, alcoholism passes off in maturer life. (3) The next bond of similarity insisted upon was the paroxysmal and impulsive character of all forms of alcoholism. It was argued that the craving was largely the result of suggestion, and that the subjects of reiterated forms of suggestion of any kind are abnormal apart from the suggestion and its results. (4) The abnormal psychical organisation of inebriates, their hereditary connection with other neurotics and their blunted sensibilities were discussed. (5) It

was held that there is in most cases a negative correlation between the desire and the tolerance of the nervous system for alcohol. (6) The distribution of alcoholism throughout the human race was held to be in accordance with what we know of the other neuroses. (7) The fact that the fundamental explanation of intoxication habits is not a physical desire of the senses, but a craving for a mental state, is sufficient to prove that the condition is a neurosis, of which the drinking habits are only one of the external manifestations.

In concluding the first part of the course of lectures the argument was restated. It was again pointed out that the theory of variation fell under the mathematical theory of chance. If, therefore, the defects and anomalies described in the preceding lectures were true variations, they could not be the result of either (1) accident or fortuitous occurrence, or (2) of the environment, and they must be hereditary. In each instance it had been shown that they were hereditary. They could not be due to the environment, for it had been shown that they were common to many of the higher species of animals as well as to man, and that they were universally distributed over the face of the globe. With regard to accident the following proposition was stated. Let us suppose a population of 60,000,000 divided into 10,000,000 families, each composed of 6 persons. If an affection occurs constantly in one person in 1,000,000 the odds against coincidence are necessarily large; but if it occurs in one parent and two children in one family, the odds against mere coincidence rise to the incalculable figure of 8,333 millions to 1. Therefore the affections dealt with are true genetic variations, and causation as ordinarily understood in medical language is inoperative to any material extent.

---



## Abstracts

### ANATOMY.

#### A NOTE ON CAJAL'S NEW METHOD FOR STAINING NEURO- (101) FIBRILLÆ.

RAMON Y CAJAL of Madrid has recently described (*Compt. Rend. de la Soc. de Biol.*, Dec. 18th, 1903) a method for the coloration of the finest elements of the central nervous system, which depends essentially on the impregnation of the tissue by a solution of silver nitrate, and its subsequent reduction by pyrogallol. An analysis giving the details has already appeared in this Review (Feb. 1904, p. 146, Abstract No. 41).

The author claims for his method a selective affinity so delicate that it stains not merely the neurofibrillæ of Bethe, but also secondary filaments (*neurofibrilles secondaires*), unnoticed by the latter. They unite the primary neurofibrillæ to each other and form a complicated network in the interior of the cell.

Weiss (*Compt. Rend. Soc. Biol.*, Jan. 1, 1904) claims to have already demonstrated this finer network of secondary filaments, not merely in the cell but in the axis-cylinder (*Journ. de Phys. et de Path. Gén.*, 1903). Thus where Bethe, Apathy, Monckeberg and others see a parallel arrangement of continuous fibrillæ, Cajal and Weiss demonstrate a veritable network.

At the March meeting of the Neurological Society of Paris, M. Azoulay exhibited some sections stained by Cajal's method. In some from the spinal cord of the rabbit, the neuro-fibrillary network in the cells was admirably shown. One from the ventral ganglia of the leech showed this network to be intensely complex, and made it difficult to comprehend Apathy's division of the filaments in the unipolar expansion into afferent and efferent groups. Further preparations demonstrating the basket cells of Parkinje clearly indicated that there was merely contiguity, not continuity, between the pericellular filaments and the nerve cell. The relation was essentially only one of contact (*Proc. Soc. Neur. in Rev. Neur.*, March 30, 1904).

In the discussion which followed, Prof. Déjérine gave the weight of his authority to the support of the now classic hypothesis of Waldeyer. The attacks on the neurone theory have always been histological: no anatomico-pathological objection of any importance has ever been advanced, and this fact in itself constitutes an argument for the ordinary view of considerable importance (Déjérine, *Rev. Neur.*, March 15, 1904).

Cajal has just published three modifications of his original procedure (*Compt. Rend. Soc. Biol.*, March 4, 1904).

## 1. To stain the myelinated axis-cylinders.

There is practically no change from the formula already published (see above), except that fine sodium sulphate ( $\frac{1}{4}$ - $\frac{1}{2}$  gramme) is added to the pyrogallic acid solution. Oil of bergamot or of cloves must not be employed to clear: it makes the impregnation pale. Carbol-xylol is recommended. Should sections from the middle of the original piece be too transparent, the coloration can be strengthened by a gold method.

Bathe the sections in the following solution—

Ammonium sulpho-cyanide	.	.	.	3 grammes.
Sodium hypo-sulphite	.	.	.	3 grammes.
Distilled water	.	.	.	100 grammes.

Just before employing, add a few drops of a 1 per cent. solution of gold chloride. Wash in distilled water.

## 2. For non-myelinated axis-cylinders, and for neuro-fibrillæ.

1. Pieces  $3\frac{1}{2}$  millimetres thick, at most.

2. Harden in—

Alcohol 96 per cent.	.	.	.	100 c.c.	} 24 hours.
Ammonia	.	.	.	a few drops—1 c.c.	

3. Wash in distilled water a few minutes.

4. Immersion in a  $1\frac{1}{2}$  per cent. solution of Aq NO<sub>3</sub> for 3-5 days in the stove (30-35° C.).

5. Reduce in—

Formol	.	.	.	5 c.c.	} 24 hours.
Acid pyrogallic	.	.	.	2 grammes	
Distilled water	.	.	.	100 c.c.	

6. Wash in distilled water a few seconds.

7. Dehydrate gradually (alcohol)—celloidin—cut—mount.

The section may be strengthened, as above, with gold chloride.

The pictures presented by this method beside staining ordinary fibres show the extraordinary richness (*e.g.* in the spinal cord) of those of the non-medullated variety.

3. For the terminations of nerve fibres.

The pieces are hardened in—

Commercial formol	.	.	.	25 c.c.	} 24 hours.
Distilled water	.	.	.	100 c.c.	
Ammonia	.	.	.	a few drops—1 c.c.	

They are then washed in running water for 6 to 12 hours and the technique of No. 2 is continued.

For pericellular plexuses, etc., this last method of procedure is recommended.

By the aid of his new technique, Cajal has been able to show that the network of fibrillæ in the nerve cell may undergo various modifications under varying conditions. In dogs and rabbits suffering from rabies, and consequently paralysed, he found a shrinking and a thickening of the network, a condition also seen in very young animals (rabbit, dog) and in the nerve cells of the hibernating lizard as shown by his assistant, Tello. On the other hand, in lizards rendered active by heat and then killed, the network in question was thinned and extended. It is therefore too absolute to say that the intracellular fibrillary plexus is unalterable. It may vary under given physiological and pathological conditions (*Compt. Rend. Soc. Biol.*, March 4, 1904, p. 372). The discovery is one which may well be found to have considerable bearing on current physiological teaching.

S. A. KINNIER WILSON.

## PHYSIOLOGY.

**CONTRIBUTIONS TO THE PHYSIOLOGY OF THE LUNGS.**  
(102) **PART II. ON THE INNERVATION OF THE PULMONARY BLOOD-VESSELS; AND SOME OBSERVATIONS ON THE ACTION OF SUPRARENAL EXTRACT.** T. G. BRODIE and W. E. DIXON, *Journ. of Physiol.*, Vol. xxx. p. 476.

IN this paper the authors describe a method by which variations in the calibre of arterioles are determined by recording variations in the rate of flow of blood through the vessels of isolated organs when these are perfused at constant pressure. The animals used were dogs, cats and rabbits, and the organs perfused were the limbs, intestines, kidneys and lungs.

In the case of the limbs and small intestines, organs known to be supplied by vaso-constrictor fibres, stimulation of the vaso-motor nerves during the course of a perfusion, even two hours after the death of the animal, always caused a constriction of the vessels. With the lung, on the other hand, no effect was obtained on stimulating any of the nerve fibres which pass to it either from the sympathetic or from the vagus.

Adrenalin, pilocarpine, muscarine and barium chloride when added to the blood perfused through the intestines or limbs all caused constriction of the vessels, but in the case of the lung, barium chloride alone caused constriction while the others produced either no change at all or dilatation. Barium chloride is known to act directly on the non-striped muscle fibres of the blood-vessels, while pilocarpine and muscarine are generally con-

sidered to act by stimulating nerve endings; and the authors produce very strong arguments to prove that adrenalin, which is much more powerful than any of the other drugs, also acts on nerve endings, and not on muscle fibres, as stated by Oliver and Schäfer.

If the vaso-constrictor nerve endings of the limbs or intestines be paralysed by apocodeine or curare, the constrictions produced by adrenalin, pilocarpine and muscarine are usually abolished or may become converted into dilatations, *i.e.* these vessels then behave like the pulmonary vessels.

The conclusion the authors arrive at from their experiments is that the pulmonary arterioles have no vaso-motor nerve supply. They also claim to have proved that the action of adrenalin is a general test for the presence of constrictor nerves to any set of vessels.

SUTHERLAND SIMPSON.

**ON THE TRANSFORMATION OF THE LAWS OF CUTANEOUS  
(103) REFLEXES IN AFFECTIONS OF THE PYRAMIDAL  
TRACT.** J. BABINSKI, *Rev. Neurol.*, Jan. 30, 1904.

It is generally admitted that in organic hemiplegia the tendon reflexes are exaggerated, while the cutaneous reflexes are abolished or diminished. Van Gehuchten lays stress on this opposition in spasmodic paraplegia. Babinski brings forward some facts to show that the contrast between the behaviour of the different reflexes is not so absolute, and insists on the importance of a more accurate description of cutaneous reflexes.

He gives the case of a patient with spasmodic paraplegia, where the antagonism between the two reflexes seemed plain: the knee and Achilles jerks were exaggerated, the abdominal reflexes were absent. But on pinching the skin of the leg, or of the thigh, there was extension of the big toe and flexion of the thigh on the pelvis, more marked than in the normal individual. This exaggeration Babinski has found in many cases of organic spasmodic paraplegia. There therefore seems in such cases to be exaggeration of the cutaneous reflexes, contrary to what the examination of the abdominal reflexes gives.

Van Gehuchten, in mentioning similar facts, still maintains the law of the contrast of the behaviour of the two kinds of reflexes. He acknowledges that all the cutaneous reflexes are not abolished, but distinguishes between normal cutaneous reflexes elicited by stimulation of a definite skin surface and pathological cutaneous reflexes elicited by stimulation of the skin over the greater part of the inferior extremity. Babinski criticises this division according to the area over which the stimulation can be employed; in some normal subjects the cremasteric reflex can be

elicited on rubbing, not only the thigh, but also the leg and the foot.

The law regulating the reflexes is therefore not so simple as would appear from the earlier formula.

In some cases of spasmodic paraplegia, while stimulation of the sole of the foot, of the leg, or even of the lower half of the thigh, may produce extension of the big toe, stimulation of the upper half of the thigh, or of the skin of the abdomen, may produce marked flexion. It is not only in affections of the pyramidal system that stimulation of the skin of the abdomen may produce a different reflex movement of the toes from stimulation of the sole. In a case of sciatica, stimulation of the sole gave much less marked flexion on the affected side than on the other; whereas this was reversed on stimulating the skin of the abdomen.

Considering, therefore, the cutaneous reflexes from a wide point of view, Babiniski concludes that it is not accurate to say that, in affections of the pyramidal system, the cutaneous reflexes are diminished and in contrast with the tendon reflexes. The truth is that the laws of the cutaneous reflexes are modified in such affections.

C. MACFIE CAMPBELL.

**THE REFLEXES IN LONG DISTANCE RUNNERS; A STUDY (104) OF THE INFLUENCE OF FATIGUE UPON CERTAIN REFLEXES.** PHILIP COOMBS KNAPP and JOHN JENKS THOMAS, *Journ. of Nerv. and Ment. Dis.*, Feb. 1904, p. 94.

THESE observations were carried out on the competitors for the "Marathon Race" at Boston, an annual event for the last seven years.

The race, extending over a course of about twenty-four miles, had its origin in the feat of Eucles, the soldier who is reputed to have run in full armour from Marathon to Athens with the news of the great victory, and to have dropped dead in the market-place while telling the news. In the revival of the Olympic games at Athens in 1896, this race was included. The Boston race is over a course with several hills, and is, needless to say, a great test of endurance and most fatiguing to the competitors.

This last year 48 candidates were examined before and 41 after the race with regard to their plantar reflex, Achilles reflex, knee-jerk, front tap contraction, ankle clonus and patellar clonus.

Of the 48 men examined before the race, in 26 there was a very marked knee-jerk and also a patellar-jerk, in 18 there was a front tap contraction in addition. The Achilles-jerk was present in all. In only one case was it necessary to use reinforcement. Ankle and patellar clonus were absent in all. In most cases the knee-jerk was exaggerated, due probably to excitement.

In 5 cases there was no plantar reflex, but it was difficult to test this accurately, as it was necessary to avoid any possible injury to the soles of the feet.

After the race 41 men were examined. In 26 the knee-jerk was diminished, in 4 a front tap contraction not elicited before was got along with exaggeration of the knee-jerk. In no case was there clonus. The knee-jerk was absolutely lost in 3 cases, in other 3 it was lost on one side and only got with reinforcement on the other. In 5 cases the knee-jerk required reinforcement to obtain it at all, and in other 5 the knee-jerks were unequal on the two sides. The ankle-jerk was also diminished as a rule, in 5 cases it was lost, and in 2 lost on one side. The loss of knee and ankle jerks did not necessarily co-exist in the same case.

The patellar-jerk, got in 44 before the race, was only obtained in 17 after it; and the front tap contraction, got in 18 before the race, was only obtained in 9 after it. On the other hand, the plantar reflex was increased in most cases after the race, definitely in 20, diminished in 5, and in 12 no change was noted. In 5 men who showed no plantar reflex before the race, 4 possessed a definite reflex after it. There was no change in connection with special exhaustion in some competitors as compared with the others. A Babinsky reflex was not obtained in any case.

The authors consider that exhaustion of the reflex centres may explain the diminution noted after the race, but it is more difficult to offer any explanation to account for the increase of the plantar reflex.

ROBERT A. FLEMING.

**THE MYOGRAPHIC AND THE ERGOGRAPHIC CURVE.** E. BEL-  
(105) MONDO, *Riv. di Patol. nerv. e ment.*, Feb. 1904.

THIS paper is a reply to Dr Z. Treves's criticism in the *Riv. di Patol. nerv. e ment.*, Jan. 1904, on the two studies by Dr L. Lugiato, published in the same paper (Sept. and Dec. 1903).

Treves's principal thesis is as follows:—Since the method by which the "ergographic curve" is obtained neither is, nor can be, rigorously isotonic, and since in the ergograph the muscles act on the writing apparatus, not directly, but through bony levers, and without our being able to exclude the influence of other movements, often variable and not always exactly calculable, it cannot be admitted that the curve of an ergographic movement, by whatsoever ergograph it is registered, can ever correspond even approximately to the curve of muscular contraction. This has not been faithfully drawn save in the common isotonic myograph; and Dr Lugiato's tracings not only are not comparable (as he believes) to those of a myographic curve, but do not have even the most dis-

tant connection with those which would correspond to a muscular contraction pure and simple.

To this criticism Professor Belmondo replies by defending the employment of analogy in scientific investigations, and by pointing out that Dr Lugiato definitely recognised that the parallel between his tracings and those obtained by the common myograph was conditioned by the difference of the experimental circumstances.

He objects, moreover, to the establishment of the isotonic myogram as paradigm of a typical muscular contraction, seeing that all we know of an isotonic contraction has been discovered by experiments on animals—and on animals in such artificial and unnatural conditions as to give even in their case little idea of the nature of voluntary muscular contraction in normal conditions. Nor is it possible to infer the laws which regulate muscular contraction in man from a study of the isotonic myograms of animals, seeing that the form of the curve varies according to the kind of animal in which it is studied. Rollett, for example, by his experiments on the abductor muscle of the little finger, finds muscular contraction in man characterised by an inactivity which differentiates it greatly from that of the animals commonly experimented on, and assimilates it rather to that of the bat. Other differences have been demonstrated by Fick and Grützner, Rollett, Schenck and Liehr, Lohmann, Novi and his pupils. From these results it follows that if we wish to learn the laws of muscular contraction in man, we must study them only and always in ourselves; and since the rigorously isotonic method has not yet been rendered applicable to man, we must make use of instruments which are based on other principles, or rather of those (such as the ergograph) which, although theoretically classified among isotonic instruments, do not in reality attain completely to their scope.

Now the ordinary instruments based on the isometric method (such as the various types of *Spannungszeiger*), and the various proposed forms of dynamograph, exaggerate enormously the slightest displacement of the bony extremity; in the latter instrument, moreover, the imperfect application of the muscular force to the handles—not to speak of the fact that the group of muscles brought into action is such as to defy all analysis—presents grave obstacles to any exact investigation.

The ergograph, on the other hand, in spite of the defects pointed out by Lugiato, can be used to study the whole course of muscular contraction, whether voluntary or induced by electric stimuli.

Treves's contention that the ergographic curve is a representation of the successive tensions of the muscle transmitted to the levers has never been disputed; it is evident from the admitted fact that muscular contraction in living animals is never isotonic. But Lugiato, for the first time, has given a clear graphic demon-

stration of the fact that the finger in the ergograph does not perform equal works in equal times, but that the work increases in proportion to the flexion of the finger; and his results obtained by aid of the little instrument devised and described by him (p. 530 *et seq.*), he has been able to confirm by means of trigonometrical calculation.

Any recognised defect of the ergograph may be avoided in one or another series of experiments. Thus the psychic influence of the subject—which, however, may itself be a subject of experiment—may be eliminated by the adoption of the electric current as stimulus; irregularity of tension by the contractions being executed according to a slow and regular rhythm both in flexion and in extension, or by the employment of a very small weight. The most difficult error to eliminate in the original ergograph of Mosso is certainly that depending on the mode of fixation of the moving finger, and the method of applying the loop to the finger. In these respects particularly the modifications introduced by Treves are a decided improvement; and indeed in such a study of ergographic curve as that undertaken by us, his instrument might have given more valuable results than Mosso's original apparatus which we adopted.

Nevertheless, when the causes of error are known and variations which may be influenced by them disregarded, and when the same change in an otherwise constant graph always follows upon the introduction of the same new experimental condition, the change being verified by experiments on other subjects, surely results thus obtained—as they have been by Professor Lugiato—may be added with confidence to the assured body of scientific knowledge.

MARGARET DRUMMOND.

## PSYCHOLOGY.

### THE RELATION OF THE NERVOUS SYSTEM TO THE MIND.

(106) P. KRONTHAL, *Neurolog. Centralbl.*, Feb. 15, 1904, S. 154.

THE author seeks first to obtain a clear idea of the relations between nervous system and mind by studying the lower animal organisms, and then tries whether our knowledge of ascertained facts from physiology and pathology can be naturally and easily explained by this idea.

Many unicellular organisms, such as the rhizopods and the higher ciliates and flagellates, show within the same cell a distinction between sensory and motor apparatus, between which the impulse must be conveyed directly through the cell-substance; and in some low multicellular organisms, devoid of nervous system,



the same principle is seen. Since, therefore, sensation is a part of mind, the latter cannot be indissolubly connected with a nervous system. In the higher organisms the nerve-cell is interposed between the sensory and motor apparatus, but the methods of Apáthy and Bethe show that the fibres neither begin nor end in the cells, but merely traverse them, and convey impulses from the sensory to the motor cells. In these, however, stimulation of one sensory cell throws many motor cells into action, and this must be the work of the nerve-cell. The nervous system is therefore merely a mechanism for the transmission of stimuli, and the assumption of central stimuli started by the nerve-cell must be an error. The more cells respond to a stimulus, the more highly developed the nervous system; and the more highly developed the nervous system the higher the mind, which is therefore directly proportional to the sum of the reflexes. It follows that "*what we call mind has no causal connection with the reflexes, but is the sum of the reflexes themselves.*" The metazoon most highly developed mentally is that in which a stimulus applied to one cell is transmitted to the largest possible number of other cells: the height of the mental development therefore depends, not on the number of the elementary organisms composing the individual, but upon the intimacy with which these are placed in impulse-conducting connection; and since the connections take place in the grey matter, this means that mental development is proportional to the amount of grey matter. When the nerve-fibres enter the grey they lose their insulating medulla, and form a fine meshwork of still isolated threads, in which certain "viscous drops," the leucocytes, become entangled. The nerve-cells are the product of coalescence of the leucocytes, which destroy the isolation of the fibres around which they flow, and hence enable a stimulus to pass from one fibre to several. The connecting fibres, the tracts, in the brain lie generally in corresponding positions, and the leucocytes which join them for the most part join the same tracts, so that the sum-of-reflexes, or mind, of different individuals has a general resemblance, divergences being explained in the same way as are bodily variations.

In infants, every stimulus produces a movement, but in later life the movement is often suppressed as the result of memory; but memory is also reflex, since it is called into action by a stimulus.

The presence of large numbers of leucocytes in the brain is argued partly from the morphological appearance of many brain-cells, and partly from the number and fineness of the cerebral vessels, and the facility, therefore, with which diapedesis could occur. It is also borne out by experiment. Methylene blue in the blood is taken up by the leucocytes. Only some of the nerve-cells are stained by the *intra vitam* method. These are therefore

nerve-cells which have come into existence as such during the experiment.

When the sum-of-reflexes is large, as in man, a small deficit produces an inappreciable effect on the mind, and therefore a diffuse disorder of the central nervous system is required to produce mental disorder. When numerous sensitive cells react too quickly we have mania—an increase of the sum-of-reflexes takes place. When such react too slowly we have melancholia; and when their reaction is irregular, hysteria. Other diseases are due to affection of the conducting tracts in large numbers. Thus Korsakoff's disease is caused by affection of numerous peripheral paths, general paralysis by that of many central paths.

If mind is the sum-of-reflexes, the child must develop mentally in proportion as the paths of conduction develop, and Flechsig has shown that the nerve-tracts do not develop simultaneously. It also follows that, as we go up in the animal scale, more and more paths must cross, and in man all cross. Damage to the elementary organisms by fatigue or poison will make them cease to react—there will be neither reflex nor mind; hence sleep and narcosis.

The view that the nervous system is merely a mechanism for the transmission of impulses is therefore supported naturally by our knowledge; and it follows that this system possesses no controlling influence in the organism, but merely a mediating function, and that there is no seat of the mind, which is the product of each organism as a whole.

W. R. DAWSON.

**A CASE OF COLOURED AUDITION WITH OBSERVATIONS ON  
(107) THE STABILITY AND HEREDITARY TRANSMISSION  
OF PHOTISMS.** AUG. LEMAITRE, *Arch. de Psychol.*, T. iii.,  
Feb. 1904, p. 164.

COLOURED audition is a phenomenon well known to psychologists. It is characterised by the association, in certain individuals, of certain sounds with a subjective sensation of colour. In the present article, M. Lemaître narrates a very interesting, if not a unique, case of the acquisition of this peculiarity.

The subject, who may be called René, was a schoolboy, 14 years of age, who had acquired what he called his "sixth sense" at the age of 7. While staying with his nurse in the country he amused himself one July day by trying to look at the sun, after which he lay down on the grass and slept for three or four hours. He was awakened by a young herdsman, who shook him and cried "Get up." As the herdsman addressed him, he was astonished to notice against the man's chest vivid colours, appearing and disappearing with the words of the speaker. From that time to the present the phenomenon has persisted. Let him hear any

words whatever, even a low whisper, and immediately colours are visualised upon a wall, a book, a sheet of paper, etc. The form usually seen is an oval, about the size of the palm of the hand, and frequently geometric patterns are seen in relief upon the coloured surface. The colours themselves vary infinitely, not only with the persons speaking, but from moment to moment. Even the same word pronounced by the same person may be yellow to-day and blue to-morrow.

In spite of this variability, however, the general tone of voice of a speaker gives his speech at least a temporary character. For instance, when René's class-mates were asked to read the same passage aloud, the speech of one was described by him as deep green, of another as orange, of another as violet and red, and so on. Generally speaking, it may be said that the deeper and stronger the voice, the lighter is the colour.

Sometimes a word appears peculiarly impressive because the colour seen corresponds with the meaning of the word. For instance the word *blood* is much more striking when associated with the red colour of blood than when associated with yellow or blue.

The colours seen do not quite correspond to those with which René is familiar apart from audition. He says they are at once more harmonious and more glaring. One may form some idea of them by thinking of an Alpine meadow spangled with flowers.

René at one time imagined that every word had its special colour, but at the age of twelve he tried to make a sort of dictionary of colours, and then found that the colours varied. His friend, for instance, one day said *black*, and René saw *blue*. Accordingly he wrote *black is blue*. But on the following day *black was red*.

The phenomenon is frequently very annoying. For instance at the writing lesson some freedom is permitted to the pupils, and René is constantly so "blinded" by the colours on his copy-book caused by the chattering of the scholars that he is unable to form his letters properly. During dictation, on account of the projection of colours on the paper, he cannot begin to write while the sentence is being dictated, but must wait until it is finished. When walking in a crowd he sees such a succession of colours that his eyes become dazzled, and he cannot see where he is going.

It is only the human voice that produces these photisms. Other sounds, such as those produced by musical instruments or by animals, do not cause them, nor do words reproduced by the phonograph.

While it is impossible to give any explanation of this case, one may suggest that perhaps the acoustic vibrations due to the loud shout ("Get up"), acting upon a young brain asleep, but in a state

of erethism from exposure to the sun, may have spread beyond their own territory and reached the visual cortex, itself in a state of violent disturbance corresponding to the negative image of the sun, at which the child had been looking before he went to sleep.

The auditory-visual association so formed, instead of being ephemeral, persisted (why, it is difficult to say; it would be an example of the cases of *association privilégiée* described by Flournoy), but exclusively for the human voice, which would suggest for this voice a special auditory localisation in relationship with visual localisations.

II. *Stability of Photisms.*—In contrast to the above, the author narrates a case of the same affection whom he questioned four times during three years and a half as to the colours of certain words, a list of which had been prepared beforehand. During that period the colours were found to vary very little—the colours of the vowels not at all. Other cases also showing a high degree of stability are quoted from Claparède (2 cases), and from Nessler (1 case).

III. *Hereditary Transmission of Photisms.*—A boy of thirteen was found to have coloured audition, the vowels being—

a red, e white, i black, o yellow, u blue.

The child had never heard the phenomenon spoken of, and when he went home and told his mother, she said she had the same peculiarity, and the vowels bore the very same colours. The diphthongs, however, differ in the two cases. In the boy the diphthongs take the colour of the initial letter (ae=red), while in the mother the colours seem to mix, thus—

au=red and blue=violet.

ou=yellow and blue=green.

ao=red and yellow=orange.

The mother has therefore transmitted to her son not a mere general tendency to photisms, but her own peculiar synoptic formula.

W. B. DRUMMOND.

## PATHOLOGY.

(1) REMARKS ON THE WORK OF DR BIELSCHOWSKY ON  
(108) THE HISTOLOGY OF MULTIPLE SCLEROSIS. ANTON  
STRÄHUBER.

(2) THE NON-MYELINATED NERVE FIBRES IN THE POOL  
(109) OF MULTIPLE SCLEROSIS: AN ANSWER TO DR  
STRÄHUBER. MAX BIELSCHOWSKY, *Neurolog. Centralbl.*, Jan.  
16, 1904, p. 55.

THESE two papers have reference to an article published by Dr Bielschowsky in the *Neurolog. Centralbl.* of August 16, 1903, p. 770.

on the results he had obtained from investigating the central nervous system in cases of multiple sclerosis by a method of axis-cylinder impregnation dependent on the reduction of an ammoniacal silver nitrate solution by formaldehyde. An abstract of this article will be found in the November number of the *Review of Neurology* of 1903.

(1) Dr Sträuber begins by pointing out that in his paper on multiple sclerosis published in Ziegler's *Beiträge zur patholog. Anatomie*, 1903, Nr. 33, he had expressed his belief in the presence of fibres which, in addition to being non-myelinated, did not possess the axostrome stainable by his anilin blue method in the foci found in cases of disseminated sclerosis. He rejoices that his opinion should have been confirmed so speedily by the work of Dr Bielschowsky. In the second place, he draws attention to the fact that he had in the same article proposed the name axochromatin in place of axostrome, on the grounds that the substance to which it refers is probably only a constituent of, and does not represent the whole of, the material supporting and cementing the neurofibrils. This view is again upheld by the work of Dr Bielschowsky, who has described axis-cylinders which are not exposed by the axostrome stain and yet, presumably, have some perifibrillar substance of a supporting character. One can therefore no longer speak of fibres which have lost their myelin sheath as necessarily naked axis-cylinders, but merely as non-myelinated fibres.

After referring to the work of Neumann, Kaplan and Auerbach on the same subject, Dr Sträuber comes at length to his main theme, the latter forming a reiteration of his belief that the non-myelinated fibrils found in the foci of multiple sclerosis consist to a large extent of regenerating elements and do not represent merely persistent axis-cylinders as affirmed by Dr Bielschowsky. The arguments advanced to support the regenerative theory are not altogether convincing, since they presuppose the possibility of this regeneration in the first place and, in the second place, assume that the course of the fibre which has been destroyed is kept open by rows of undifferentiated protoplasmic cells, in order to explain the fact that Dr Bielschowsky has traced the axis-cylinder from the point where it loses its myelin, through the patch of disease, to its continuation as a myelinated fibre once more in healthy tissue. Again, Dr Sträuber believes that the brush-like branching of the fibrils is an expression of regeneration rather than of degeneration, and in support of this conclusion states that he has seen such branching not only within tube-like cavities, but also in positions where the two or more branches were separately walled in by interstitial tissue.

On the other hand, no adequate explanation is provided for the

fact that in older foci where regenerative signs should certainly be present, very few or no axochromatenin-containing fibres are to be seen. By means of embryological investigations on sheep, Dr Sträuber has found that the axochromatenin can never be stained before the appearance of the myelin sheath, a fact which, in conjunction with the last observation, would appear to speak against rather than for the theory of regeneration.

In conclusion, the writer expresses his entire agreement with the view that histogenetically the disease process in multiple sclerosis is an inflammatory one affecting simultaneously parenchymatous and interstitial structures, while etiologically it is of toxic origin, the poison producing its effect by way of the blood-vessels.

(2) Dr Bielschowsky confines himself to the question of the non-myelinated fibres being regenerated or persistent axons, and states that the majority of methods and the majority of workers favour the latter view. Two other authors using the impregnation method came to the same conclusion. Bartels examined four cases and demonstrated the continuity of the fibres within and without the foci, and believed that he had obtained anatomical proof of the permanence of the conducting elements. Hoffmann had used Sudan combined with Weigert's myelin stain, and had shown that the medullary sheaths were broken up with fatty change on the borders of the diseased patches in such a way as to suggest that each individual patch spread centrifugally. He contended that it was hardly possible that a toxic agent could at one and the same time favour the disintegration of the myelin sheath and the regeneration of the axis-cylinder in the same nerve fibre.

In reference to the granular cells to which Dr Sträuber attributes the duties of a quartermaster-sergeant in providing nutrition for the new fibres, Dr Bielschowsky regards them more in the light of scavengers and points out that they are to be met with in most parenchymatous degenerations of the central nervous system. The embryological results Dr Bielschowsky interprets in an opposite sense to that taken by Dr Sträuber, and he thinks one may assume that a regenerating fibre is built up in a similar way to a foetal one, the latter having been proved to receive its axochromatenin at the same time as its myelin sheath. For this reason he would regard the fibres stained with anilin blue in the disseminated foci as persistent axis-cylinders whose axochromatenin has outlasted their myelin. This condition is frequently observed in recent foci and in the borders of older foci.

Dr Bielschowsky regards the brush-like or dichotomous branching as evidence of degeneration; on the other hand he believes

that the right-angled branching is connected with the formation of new fibres. Finally, he affirms that he has no wish to deny the possibility of the regeneration of fibres in the central nervous system: on the contrary, the results which he has obtained from examining a case of syphilitic meningo-myelitis of the second dorsal segment by his impregnation method have led him to believe that regeneration does take place. Moreover, in the light of what he found in this last case he has re-examined his specimens of multiple sclerosis and has found what he considers to be a few newly-formed naked fibres coursing round the borders of diseased patches. While he agrees with Dr Strähuber that regenerated fibres are present in foci of multiple sclerosis, he emphatically denies that they are anything like equal in number to the persistent axis-cylinders. E. FARQUHAR BUZZARD.

(110) **THE NATURE OF TABES.** MILIAN, *La Syphilis*, Jan. 1904.

THIS is a very concise, accurate, and up-to-date analysis of our present knowledge on a far-reaching and all-important subject. A brief but sufficient resumé of the structure of the posterior columns of the cord is followed by an account of tabetic topography, including the histological changes characteristic of the disease. The pathogeny of tabes is discussed under two heads: interstitial and parenchymatous theories respectively.

**INTERSTITIAL.**

- A. Vascular or dystrophic theory.
  - 1. Fatty degeneration of vessel walls (Ordóñez, 1862).
  - 2. Sclerosis of vessel walls (Martin, 1881; Brault, etc.).
- B. Interstitial myelitis of posterior columns (Buzzard, Rumpf, Adamkiewicz).
- C. Inflammation of pia mater (Redlich and Obersteiner, 1892).
- D. Syphilitic meningitis (dura mater) and transverse neuritis (Nageotte, 1894).
- E. Syphilitic changes in posterior lymphatic system of the cord (Marie and Guillain, 1903).

**PARENCHYMATOUS.**

- A. Peripheral neuritis (Désjérine).
- B. Lesion in spinal ganglia.
  - 1. Anatomical (Hallopeau).
  - 2. Functional (Brissaud, De Massary).
- C. Medullary lesion.
  - 1. In column of Goll (Bourdon and Lys).
  - 2. In the "bandelettes externes" (Charcot and Pierret).

3. Root lesion (Vulpian, Schultze, Déjérine, Redlich); Neuritis of posterior roots (Thomas and Hauser).

The second part of the paper is devoted to the etiology of tabes, historical and critical. References to the literature are fairly numerous.

S. A. KINNIER WILSON.

**NERVE SUTURE AND NERVE REGENERATION.** PAUL B. (111) HENRIKSEN, *Nord. Med. Arkiv.*, Häft 2, Nr. 9, and Häft 3, Nr. 16, 1903.

THE paper opens with a fairly full historical account of regeneration of nerves, according to the different theories, and a description of the degenerative changes occurring in the peripheral end of a divided nerve.

A valuable series of cases of nerve suture, both primary and secondary, follows. These are taken from hospital practice. They go to show the great rapidity with which sensory impulses can, at least in a limited degree, travel along the reunited nerves. One case out of the twelve recorded is specially interesting. A girl, æt. 15, coming in contact with a circular knife, was severely cut in the front of the forearm. The divided nerves were at once sutured, and in three days she had slightly regained sensation in the realms of the injured nerves, while she could move the affected muscles in twenty-two days. As a whole, one is struck in reading the cases recorded, by the very rapid regain of conductivity, both as regards sensory and also motor impulses. Motor power preceded the return of normal electrical reactions (in the nerve).

These cases induced the author to attempt to answer the following problems by experimental work.

1. Can primary suture occur without degeneration of the peripheral segment?

Nineteen experiments were carried out on rabbits, the peroneal nerve being cut and accurately sutured—of course with antiseptic precautions. In every case there was undoubted degeneration of the nerve fibres in the peripheral segment, and the electrical reactions suggested that no primary union without degeneration had occurred. Electrical excitability became normal in fifty-five or fifty-six days.

2. Can a degenerated nerve conduct impulses?

Such conduction might be due to either the degenerated nerve actually conducting impulses, or else conduction might be possible during and presumably on account of the early commencement of regeneration. A second series of experiments was undertaken on rabbits in which both peroneal nerves were cut. On the one side



one end of the divided nerve was sutured to the skin of the wound, on the other side the peroneal nerve was carefully sutured so as to ensure primary reunion. The animals were killed at dates varying from 12 to 59 days. The muscles on the two sides, supplied by the peroneal nerves, were carefully dissected out and weighed, and it was noted that before the 36th day there was little difference between the weights on the two sides, but after that period the healed side gained rapidly, and this coincided with the return of movement in increasing degree. Electrical irritability of the nerve (presumably) did not return until after motor power had reappeared.

The author examined the central and peripheral ends of the nerves and also the site of reunion in those nerves where reunion had occurred. Various fixing agents were used, Müller's fluid, Flemming's solution, Marchi, and formalin, and the nerves were mounted on wood and cut in celloidin. The staining methods employed were Weigert's hæmatoxylin; osmic acid and saffranin; Van Gieson's fuchsin, picric acid, and hæmatoxylin; and Stræbe's anilin blue and saffranin. The author considers the last uncertain and specially commends Weigert's method.

In the central ends of the nerves the following was noted, and it constitutes practically the author's views on regeneration, whether in the central or peripheral ends of the divided nerve.

There are, he believes, axis-cylinder nuclei which lie inside the myelin sheath of the nerve-fibre. These are not well seen until the myelin has been removed, as in degeneration, and although they exist in normal nerve-fibres are often unnoticed. He does not say whether these nuclei lie on, or are an integral part of the axis-cylinder, but they evidently constitute the nucleus or centre of what may be called the segmental nerve-cell. There may be two of these, however, in each segment, or in other words, between two nodes of Ranvier, one at either end. These may be seen in both longitudinal and transverse sections. The nuclei divide and so form new axis-cylinder segments, and have formed round them their myelin sheath and presumably also the neurilemma sheath. No mention is made of the rôle which the neurilemma nuclei play. It should be stated that the existence of nuclei lying on the axis-cylinders has been previously asserted by Schiff, Bruch, Adamkiewicz, and others.

The author concludes that regeneration occurs in the peripheral segment in the way just described, that degeneration occurs in the peripheral segment, and that regeneration starts so soon that even by the fourth day, if not earlier, sensory impulses may be carried along the nerve. He thinks that immediately after both primary and secondary suture there is often better conduction of sensations than there is after a few days have elapsed, and that improve-

ment is then much slower than the early return of sensation would render likely.

A damaged nerve is a bad conductor, and the longer the piece of nerve damaged the greater will be the difficulty in the way of return of conducting power, and the greater the degree of regeneration before we get sensory impulses passing upwards from the affected area. The further from the periphery the lesion is, the longer time it will take before impulses, and especially motor, are carried. Nerves heal rapidly, but in all cases suturing should be carried out, and in performing secondary suture the cicatrix which has prevented reunion should be removed, and also the peripheral end should have the usual pointed part removed.

The paper is illustrated by a number of photographs, which add much to its interest; and although we may not be able to accept the conclusions or agree with the mode in which regeneration is said to occur, we cannot help appreciating the work, and especially the valuable contribution to the published cases of nerve suture in the human subject.

ROBERT A. FLEMING.

**AN EXPERIMENTAL STUDY ON THE REGENERATION OF  
(112) POSTERIOR SPINAL ROOTS.** WILLIAM G. SPILLER and  
CHARLES H. FRAZIER, *Univ. of Pennsylvania Med. Bull.*, June  
1903.

THIS paper describes experiments, of which only one proved successful, undertaken with the object of testing whether regeneration could occur in the intermedullary portion of the posterior spinal nerve roots after section had been performed in a way likely to conduce to such regeneration. Dogs were selected for the purpose of experiment, laminectomy of two or three vertebræ being performed, and the posterior nerve roots being divided after either sutures had been introduced, or at least care taken to ensure accurate apposition of the divided ends. All the dogs died except one which lived ten months, when it was killed. A small sinus remained open for a week after the operation, a point which perhaps renders less valuable the results of the experiment.

The authors found no evidence of any regeneration having occurred on the affected side. This is contrary to the results of Bikele and Stræbe, and the authors briefly refer to the denial by Münzer of any "peripheral" regeneration in the distal end of a divided nerve in antagonism to the work of Bethe and many others.

They therefore desire more evidence before accepting the "peripheral" theory of regeneration.

ROBERT A. FLEMING.

**NOTE ON THE ULTIMATE FATE OF VENTRAL CORNUAL  
(113) CELLS AFTER SECTION OF A NUMBER OF POSTERIOR  
ROOTS. W. B. WARRINGTON, *Journ. Physiol.*, Vol. xxx. Nos.  
5 and 6, p. 503.**

IN this note Warrington gives the results of an examination of the condition of the ventral cornual cells in two dogs, in which Sherrington had divided a number of posterior roots.

In the first, six roots, viz., 4th to 9th post-thoracic, were divided and a homolateral section of the cord high up. The animal was killed in 150 days. The 6th, 7th and 8th segments were examined; in a given number of sections 914 cells were counted on the normal side, 781 on the side of the lesion. The diminution was about equally distributed. In Clarke's column also there was considerable atrophy of cells on the side on which the roots were cut. In the second experiment nine roots, viz., 2nd to 10th post-thoracic, were cut, and also a homolateral section of the cord high up. The animal was killed in 223 days. The 4th to 9th segments were examined. A count gave 2012 cells in the normal side, 1388 on the side of the lesion. The maximum intensity of the diminution was noted in the 6th segment, but in the 7th and 8th also considerable diminution was found. Clarke's column also suffered severely.

In both observations it was found that the posterior-external group of cells was chiefly affected. The writer notices his previous results on this subject and those of Braeunig (reference in this Journal, 1903). He considers that, experimentally at least, the section of fibres of exogenous origin is capable of causing an amyotrophy.

AUTHOR'S ABSTRACT.

**CLINICAL NEUROLOGY.**

**A CONTRIBUTION TO THE CLINICAL AND EXPERIMENTAL  
(114) STUDY OF CEREBRO-SPINAL MENINGITIS DUE TO THE  
DIPLOCOCCUS OF WEICHELBAUM. E. RIST and A. PARIS,  
*Arch. Gén. de Méd.*, Feb. 23, 1904.**

IN this paper three cases of cerebro-spinal meningitis are described, and along with these the results of some experiments carried out with a view to explaining the diversity of symptoms met with in this disease.

The first case was that of a child æt. 11. The illness was ushered in with symptoms of gastro-intestinal catarrh accompanied by an extensive purpuric rash. The child recovered from these

initial troubles, but three weeks later began to suffer from hæmaturia. This in turn passed off, but an acute nephritis, complicated by diarrhoea and a rise of temperature supervened. This condition remained for about fourteen days, and then all the signs of cerebro-spinal meningitis were superadded, and the child died three days later. A lumbar puncture was performed three days before death, but no fluid was obtained. The autopsy revealed a purulent cerebro-spinal meningitis. The pus was mainly composed of polymorpho-nuclear cells. A diplococcus staining with Gram's method was found in many of these cells, and when grown on blood agar a pure culture of the diplococcus of Weichselbaum was obtained. The kidneys were in the "large white" stage and no organisms were detected in them.

The second case occurred in a child æt. 3, who was admitted suffering from cerebro-spinal meningitis. Lumbar puncture was performed twice and on both occasions pure cultures of the diplococcus of Weichselbaum were grown from the fluid obtained. The child recovered completely but was brought back again two months later with all the signs of the disease well marked and died soon after admission. A lumbar puncture made at this time only showed a great number of lymphocytes, no diplococci being found. No autopsy was permitted.

The third was an acute case, which died within forty-eight hours of admission. Pure cultures of the diplococcus of Weichselbaum were grown both from the fluid obtained by lumbar puncture and from the pus which was found at the post-mortem.

The authors consider that the clinical features exhibited by these cases were due to the action of the specific organism described by Weichselbaum. As the result of many inoculation experiments which they performed, they have verified some previous observations and drawn some fresh conclusions. Their results point to the following conclusions:—

1. That the diplococcus is almost always found intra-cellular.
2. That it is hard to find in chronic cases.
3. That it may be found by culture when not seen microscopically.
4. That in cultures it may be alone, in pairs, in tetrads, and in groups, but not in chains.
5. That its virulence cannot be increased by passage through animals.
6. That the effects produced by the organism are due to its toxic action and not due to its multiplication or septicæmic action.

7. That the toxin is very slightly soluble.
8. That the toxin is very intimately connected with the body of the organism.

T. GRAINGER STEWART.

OTTO SCHULZE & Co. Successors  
K. Williams & Morgan, 20



South Frederick Street, Edinburgh.

Price Lists.

Manual of Psychiatry.

Dr. E. S. (Lecturer of Neurology at Vienna University). *Handbuch der Psychiatrie*

Psychiatrie, 5vo. Vienna, 2/-

*Lehrbuch der Psychiatrie*, Valentin del sistema nervoso. Parts 14-26. 4to. 1/- each.

*Lehrbuch der Psychiatrie*, Die Fieber psychische, with illustr. 8vo. 1904. 1/3.

*Lehrbuch der Psychiatrie*, Dr. K. Verhütung der Geisteskrankheiten, 8vo. 1904. 1/-.



**A CASE OF CHRONIC INTERNAL HYDROCEPHALUS IN A**  
(115) **YOUTH.** E. E. SOUTHARD and W. F. ROBERTS, *Journ. Nerv. and Ment. Dis.*, Feb. 1904, p. 73.

THIS case is recorded as a piece of evidence against the existence of the so-called idiopathic form of chronic internal hydrocephalus. The patient was a boy of seventeen, who was admitted to the Boston City Hospital in August 1902, with the history that ten years before he had suffered from a compound fracture of the left side of the vertex of the skull. Seven and a half years before admission he had received a severe contusion on the occiput from the kick of a horse. He had made a perfect recovery from both accidents. During the seven months preceding his entrance he had developed the following symptoms: vertigo; attacks of unconsciousness lasting two minutes, and accompanied by fits of an epileptic nature; cerebral vomiting, constant headache, blindness, anosmia, and ataxia. Double optic neuritis had been noted five months previously. The left knee-jerk was absent. A cerebellar lesion was diagnosed, but on trephining nothing was found except evidence of great intra-cranial tension. The right ventricle was tapped and several ounces of clear fluid withdrawn. This temporarily relieved the symptoms, but seventeen days later he had an attack of vomiting and rise of temperature, accompanied by occasional clonic spasm of the right arm. The right ventricle was again tapped, but the boy died next morning.

A post-mortem examination was made, and there was found a widespread chronic leptomeningitis both vertical and basal—most marked at the base, especially round the olfactory nerves, the optic chiasma and the circle of Willis. The brain substance showed the effects of great increase of tension, but was otherwise normal. The ventricles were much distended. Marked adhesions were present between the medulla and the hemispheres of the cerebellum. Both choroid plexuses presented psammomata, and the upper surface of the right choroid plexus was overgrown with connective tissue. The right anterior choroid artery showed a well-marked yellowish patch of arterio-sclerosis, diminishing the calibre of the artery by two-thirds.

The authors offer as a possible interpretation of the case that fluid effusion had occurred, the result of lowered pressure in the choroid area due to the focal sclerosis of the right anterior choroid artery. Such sclerosis is not uncommon in old age, and the fact that hydrocephalus does not occur in such cases is explained by the absence of chronic changes causing obstruction at the lymph outlet.

The writers doubt the existence of any form of internal

hydrocephalus as a separate disease, and would regard all as merely partial phenomena occurring in conditions of widely different pathology. After disposing of acute internal hydrocephalus and congenital internal hydrocephalus, they come to the conclusion that only two forms of chronic internal hydrocephalus are tenable as separate entities—one due to an inflammatory process, and the other to venous stasis. With reference to the inflammatory form, they contend that cases are gradually being recorded to give a satisfactory structural explanation of the condition, and they cannot believe that venous stasis alone will produce hydrocephalus. A summary is given of seven cases collected by Schulze. Chronic inflammation of the plexuses or meninges is the pathological condition usually recorded in such cases. The authors hold that there is an *a priori* doubt as to whether chronic inflammation can alone cause hydrocephalus, and that even if it could, the supporters of hydrocephalus as an entity must establish chronic inflammation as an entity.

In conclusion, reference is made to the work of Joslin on the subject, and to similar cases recorded by Newman and by Trendelenburg. A list of references is given.

HENRY J. DUNBAR.

**ACUTE OPTIC NEUROMYELITIS.** E. BRISSAUD and BRÉCY, *Rev. (116) Neurol.*, Jan. 30, 1904.

THE authors record a case of acute myelitis with optic neuritis which ran an exceedingly rapid course: there have been published only about twenty-five cases of this combination of symptoms, to which Devic has given the name of acute optic neuromyelitis.

The patient, a lad of sixteen, was suddenly seized one night with violent frontal headache; next day he suffered less, but his sight was dim, and two days after the headache he felt weakness in his inferior extremities and could only walk with difficulty. Examined on the fourth day he presented congestion of both discs; right pupil large and not reacting to light, left pupil large and reacting both to light and accommodation. On the fifth day walking became impossible; knee-jerks slightly exaggerated, Babinski's sign present on both sides; complete anæsthesia to touch and pain over the lower half of the body, reaching up to the tenth rib. Speech then became affected; divergent strabismus of left eye, vertical nystagmus, incontinence of urine; death thirteen days from the onset of symptoms.

On microscopical examination of the cord the perivascular sheaths in the cervical and dorsal regions were full of large epithelioid cells. The substance of the cord in these regions was normal.



In the medulla the lesions were more diffuse, the anterior and anterolateral horns being affected as well as the posterior columns: this condition was continued up as far as the middle of the olives, when the lesion was again confined to the perivascular sheaths. The nerve-roots and peripheral nerves showed no appreciable lesion. The optic nerve showed proliferation of the connective tissue, increase of neuroglial nuclei with complete disappearance of the nerve fibres; the vessels were normal.

The authors attribute the anæsthesia of the lower half of the body to a lesion in the bulb, limited to the conducting fibres from that region. They consider the cells in the perivascular sheath to be due to proliferation of the normal cells of this sheath, which take up phagocytic action. The simultaneous affection of cord and optic nerve would be simply due to an infection being localised in several distinct points.

C. MACFIE CAMPBELL.

**ON PROGRESSIVE SPINAL MUSCULAR ATROPHY IN EARLY (117) CHILDHOOD.** GOTTFRIED VON RITTER, *Jahrb. f. Kinderheilk.*, Feb. 1, 1904, S. 224.

Two cases are reported of progressive muscular atrophy in early infancy in which there was a spinal lesion and which conformed to the type described by J. Hoffmann, Bruns and others. As the author points out, this very rare disease is to be regarded as a new species of spinal amyotrophy on the following grounds:—

1. *Its being hereditary or occurring in families.*—This has not hitherto been a recognised feature in spinal amyotrophies. In these cases, however, though generally present, it is not invariably so, a number of the published cases having been sporadic in origin.

2. *The early age of the patients.*—Most neurologists used to deny that disease of this kind occurred in young children. Yet in nearly all the published accounts of these cases the disease began within the first year of life.

3. *The rapid and peculiar course of the disease and its pathological anatomy.*—The adult disease is a peculiarly chronic affection, lasting ten or fifteen years, or even longer, but in these infantile cases from one to two years is the limit of duration in the majority of instances. In the adult type the paralysis begins in the hands and spreads later to the shoulders and trunk, affecting the lower limbs little if at all. In these infantile cases the muscles about the pelvis and lower extremities are first affected, and the trunk, neck and upper limbs are implicated at a later stage.

The two cases recorded correspond closely in symptoms and in

their pathological anatomy with those published by former writers. There was, however, more extensive and extreme lipomatosis of the muscles. This condition is regarded by von Ritter as merely secondary to the spinal lesion. JOHN THOMSON.

**ON THE SYMPTOMS AND PATHOLOGY OF APOPLETIFORM**  
(118) **BULBAR PARALYSIS.** BREUER und MARBURG, *Arch. aus dem. Neurolog. Institut. an der Universität Wien*, H. ix., 1903, p. 181.

THIS paper includes the full clinical and pathological study of two interesting cases of acute bulbar palsy due to gross vascular lesions, and a careful consideration of part of the literature on the subject.

In the first case, a man of 57, there was clinical evidence of extreme arterio-sclerosis. Two months previous to the onset of the fatal illness, there was a slight attack—headache, vomiting, vertigo, and transitory difficulty in swallowing—from which he rapidly recovered. Eight days previous to death he suddenly and without warning collapsed, without loss of consciousness. The old symptoms, with greater difficulty in swallowing, hoarse voice, transient diplopia and paræsthesia of the left side of the face, quickly followed. When he came under observation he staggered in walking and tended to fall to the left. The left limbs were ataxic, the left side of the face and the right side of the trunk and the right extremities were hypoalgesic and the muscle sense was diminished in the left. There was also bilateral weakness of the palate with marked affection of deglutition and some aphonia.

The autopsy confirmed the diagnosis of arterio-sclerosis and thrombosis of the left vertebral artery, while the microscopical examination of the medulla brought to light the existence of two foci of softening, an older one which extended from the anterior third of the inferior olive to the level of the facial nerve involving the lateral two-thirds of the formatio reticularis, sparing the greater part of the olive and the structures ventral to it and not reaching the floor of the ventricles. The structures destroyed included the proximal two thirds of the nucleus ambiguus, the proximal half of the vagus fibres, and the greater part of the glosso-pharyngeus, the spinal roots of the glosso-pharyngeus and acusticus, and that of the trigeminus caudal to the facial nerve, the median part of the corpus restiforme and part of the ventral cochlear nucleus.

The second focus included the superior olive, that portion of the substantia reticularis lying between it and the spinal fifth root and the structures contained therein.

The second case was a man of 38, a subject of cardiac trouble. In the course of a few hours the symptoms developed, viz: weakness of the left side of the face, difficulty in swallowing and regurgitation of fluids through the nose, affection of hearing, transient diplopia, weakness and clumsiness of the left hand and staggering gait. When he came under observation the day before death, which occurred on the 8th day of the illness, he staggered and reeled when placed on his feet and tended to fall to the left, there was some diminution of sensation on the right side of the trunk and less definite loss on the left side of the face, ataxy of the left limbs, weakness of the left side of the face and of left side of the tongue, weakness of lateral deviation of the eyes, especially to the left, bilateral palate palsy and difficulty in swallowing.

The post-mortem examination revealed thrombosis of the left vertebral and of the basilar arteries following embolism of the vertebral. Sections of the medulla showed an area of recent softening of practically the same extent as that in the first case, but there was besides a focus of softening in the upper part of the dorsal columns of the cord and their nuclei.

But from the clinical point of view the interest undoubtedly centres in the occurrence in both cases of definite symptoms of involvement of the cervical sympathetic system, on the same side as the lesion, *i.e.* ptosis, myosis, retraction of the eyeball, and in the second case vaso-motor symptoms in the face, all on the same side as the lesion. The occurrence of these associated with bulbar symptoms has recently been drawn attention to by Hoffmann, and the authors collect other cases from the literature, and show that symptoms of cervical sympathetic palsy in bulbar and pontine cases is on the same side as the lesion, while if the disease be in the cerebral hemispheres, as Nothnagel first described, it occurs on the contralateral side. The sympathetic fibres pass caudalwards from the internal capsule, decussate in the pons Varolii, and run in the dorso-medial part of the substantia reticularis into the cord.

No secondary degenerations were found in the second case owing to the recent date of the lesion. Those present in the first were carefully investigated by Marchi's method, but the results are mostly incomplete as the cord was not examined. The most important are:—

- (1) Degenerated fibres are found dorsal and ventral to and partly within the inferior olive, which at its caudal end unite into a bundle which occupies the position of Helweg's tract in the cervical cord. Their case cannot throw any light on the origin of these fibres, but after a consideration

of the literature, the authors incline to the opinion that they may take origin in Deiter's nucleus.

- (2) The fasciculi comitantes trigemini—bundles lying on the medial surface of the substantia gelatinosa Rolandii which remain intact after extra-medullary section of the trigeminous root are degenerated. Their origin and other connections are doubtful.
- (3) The inferior olives are doubly connected with the cerebellum, by tracts running in opposite directions.
- (4) There is probably a tractus nucleo-cerebellaris in connection with the cochlear nuclei.

In the attempt to correlate the clinical symptoms with the anatomical state, the staggering gait with tendency to fall to the side of the lesion and the ataxia of the homolateral limbs is ascribed to involvement of Deiter's nucleus; the bilateral palsy of the palate is due to a lesion of the middle and proximal parts of the nucleus ambiguus, whose efferent fibres in part decussate; and the motor innervation of the pharynx and larynx comes from the same nucleus.

The author's conclusions on the arterial supply of the medulla are in almost complete agreement with the classical results of Duret.

GORDON HOLMES.

**ON THE MANIFESTATIONS OF TUMOURS OF THE ROLANDIC (119) OR MOTOR REGION.** F. DURET, *Rev. de Chir.*, mars 1904.

THE author analyses the general characters of Rolandic tumours under the separate phases of spasms, convulsions, paralyses, contractures, muscular atrophy, and sensory disturbances. Concise descriptions are given of localising features in the symptoms. An excellent table is appended of sixty-four cases of Rolandic tumour where sensory troubles existed, and the exact seat of the tumour as found at the operation is indicated in a corresponding column. Numerous references to the literature (almost entirely, however, French).

S. A. KINNIER WILSON.

**PROLONGATION OF THE REACTION OF THE PUPILS TO (120) POISONS AS AN EARLY SIGN OF GENERAL PARALYSIS.** ED. TOULOUSE and CL. VURPAS, *Journ. de Neurol.*, Feb. 5, 1904, p. 52.

USING one drop of a 1 in 10,000 solution of atropine or eserine, the writers find that (1) generally the pupillary modification takes

longer to reach its maximum in general paralysis than in the normal state, and (2) the most marked difference is the prolongation of the total duration of the reaction in general paralytics—usually it is about three times as long as in normal individuals. The same slow muscular reaction is seen in striped muscle, and is indeed one of the earliest manifestations of general paralysis. The reaction of the pupil is, however, more delicate, and may be evident to the eye earlier than in the case of striped muscle, and therefore this change in the reaction of the pupil to atropine or eserine may be placed among the early signs of general paralysis.

A. W. MACKINTOSH.

**A CONTRIBUTION TO THE STUDY OF AUTO-INTOXICATION  
(121) IN EPILEPSY.** G. GUIDI, *Ann. d. Istit. Psichiat. d. R. Univ.  
di Roma*, Vol. ii., 1902-1903.

FROM a review of the recorded chemical investigations into the pathology of epilepsy, and a consideration of the present knowledge regarding the origin of the ammonium salts normally found in the urine, the author infers that the amount of ammonia in combination eliminated in the urine corresponds to the degree of acid intoxication of the individual. Granting that, as would appear from recent researches, in idiopathic epilepsy the special intoxication is due to carbamic acid, variations in the quantity of ammonium compounds in the urine ought to be an index of the degree of this intoxication. He has endeavoured to test the validity of this contention by investigations upon nine cases of epilepsy. Four of the patients were kept upon a mixed diet, and the remaining five upon a diet consisting of milk, eggs and vegetables. In the first group the average amount of ammonia excreted did not appear to exceed the normal; it was slightly increased in the second. In both groups there were very marked irregular oscillations in the amount excreted, and its sudden rise was generally associated with the occurrence of a fit. The tabulation of the observations shows that the amount is greatly increased before the occurrence of a fit, and that it remains high, or even undergoes further increase, for one or two days.

The author concludes that in cases of idiopathic epilepsy the amount of ammonium compounds excreted increases in correspondence with the accumulation of toxic substances, which, when the organism becomes surcharged with them, determine the occurrence of a fit. The continued increased excretion of these compounds is to be explained by the fact that the organism is ridding itself of the accumulated toxic substances. Afterwards

the excretion diminishes, to increase again when the accumulation of toxins recommences. The elimination of ammonium compounds has thus a definite relation to the occurrence of the fits.

W. FORD ROBERTSON.

**A CASE OF CEPHALIC TETANUS WITH FACIAL DIPLEGIA**  
(122) BOUCHAUD, *Journ. de Neurolog.*, Feb. 5, 1904, p. 41.

THE patient was a man, aged 36, an epileptic, who fell face forward on the earth during an attack, cutting the dorsum of his nose along its whole length. On the eighth day after this accident, trismus, dysphagia and bilateral paralysis of the face appeared simultaneously, and the case developed into a clear example of cephalic tetanus, with fatal issue on the sixth day in an attack of dyspnoea and suffocation. The facial paralysis was of a peripheral type, both upper and lower facial regions being completely paralysed, with the exception of slight voluntary movements of the lips. The faradic excitability of the facial muscles was normal.

The author reviews shortly the various symptoms of cephalic tetanus, but we shall deal only with the most striking feature of his case, viz., the bilateral facial paralysis. Facial paralysis is one of the most characteristic signs of this form of tetanus, but it is very rarely bilateral. It is almost always confined to one side, and this is usually the side on which the wound lies. Bouchaud finds that bilateral paralysis has been recorded in only four cases out of forty-five, and in all these cases the wound, from which the tetanus has started, has been in the *middle* of the face, as in his case—generally about the nose. In most cases the electrical reactions have been normal, as in Bouchaud's case; reaction of degeneration (Bernhardt) and increase of electrical excitability (Oliva) have been recorded. The facial paralysis often appears at the outset, at the same time as the trismus, as in Bouchaud's case, but it may precede or follow trismus. The characters are those of a peripheral facial palsy; it may, however, be limited to the lower facial muscles (Middeldorf). Spasm of the facial muscles may exist on the same side as the paralysis, or on the opposite side.

Cephalic tetanus is due to the introduction into the tissues of Nicolaïer's bacillus and its toxin, from a wound of the face, usually in the orbito-temporo-nasal region; probably bilateral facial palsy was due in Bouchaud's case to the wound being in the middle line of the face, so that the toxin could be carried along *both* fifth nerves to the bulbar centres. But there is as yet no satisfactory explanation of the existence of a state of paralysis—

it is the rule in cephalic tetanus, while it is very rare in ordinary tetanus, and it is very difficult to explain this difference. Brieger has isolated, in cultures of Nicolaïer's bacillus, six kinds of tetanic ptomaines, and one of these, tetano-toxin, has a feeble convulsive action, showing its effect chiefly by generalised muscular paralysis. It has been maintained that the facial paralysis is always preceded by spasm. If this were true, the paralysis might be attributed to the tetano-toxin. But this is not in accordance with fact; in Bouchaud's case the facial paralysis existed as such from the outset, and there was no preceding spasm.

We note that the mortality in cephalic tetanus is stated to be about 60 per cent., and in ordinary tetanus 84 per cent.

A. W. MACKINTOSH.

**A CASE OF ALEXIA.** PHILIP ZENNER, *Journ. of Nerv. and Ment.* (123) *Dis.*, Feb. 1904, p. 89.

THIS case is one of great clinical interest, although it is not strengthened by a pathological examination.

A man aged 63 had a venereal sore when 23 and a longstanding history of articular rheumatism. There was dilatation of the heart, but it was doubtful whether there was any endocarditis, and a murmur was never clearly made out. The cerebral lesion was either a cerebral embolism or endocarditis obliterans causing an area of softening in the left occipital lobe (in part) and the contiguous portion of the angular gyrus.

The clinical features were as follows. The patient was in hospital with a "heart attack," when he suddenly found he could not read. He was doubtful on which day this came on, but with this loss of ability to read he had difficulty in getting the right word which he wanted to use when speaking. The author examined the case six months after these symptoms appeared. The patient had right homonymous hemianopsia, but not complete peripheral scotoma, although it extended to the centre. In addition, the scotoma was divided into an upper absolutely blind quadrant and a lower relatively blind quadrant in each eye. The absolutely blind quadrant is larger in the left eye. Central vision is normal. The patient repeats himself and is evidently mentally weaker than normal, but understands what is said to him; he can name things shown to him as a rule but absolutely fails to read. He can find a letter asked for but cannot name a letter pointed out to him. He writes well but is somewhat faulty in spelling, said to be an old failing. There are no motor or other features in the case. There was no improvement during several months of observation.

The author concludes that the angular gyrus is probably involved, but not completely, as otherwise the phenomena would have been more marked.

ROBERT A. FLEMING.

**ON A CASE OF DYSPANTIGRAPHIA.** C. GULBENK, *Rev. Neurol.*, (124) Feb. 15, 1904.

THE author gives the case of a patient with a rare disturbance of the faculty of writing. The patient, 70 years of age, came into hospital for difficulty with his speech and paresis of the inferior facial of twenty days date. Speech was slow and embarrassed, the words badly pronounced but intelligible, no transposition of syllables, no jargonaphasia; nothing abnormal about the limbs. As to writing, the patient could write pages fluently and legibly to dictation. But if asked to copy, he could only do one or two lines and then began to misspell, and finally became illegible about the fourth line; but if again dictated to, he could immediately write without fatigue.

Traces of aphasia present on his admission soon developed, and the aphasia soon became almost complete, only a few words remaining. At this stage writing was quite impossible.

Gulbenk holds that this case shows that the truth lies between the view that our hand copies visual images stored up in the angular gyrus, and the other that our hand translates directly into graphic signs the sounds furnished by internal speech.

In writing we use at the same time visual images and these sounds. Admitting a graphic centre at the posterior extremity of the second frontal convolution Gulbenk explains the symptoms of the case by postulating a functional ischæmia, leaving intact the connection of auditory (temporal lobe) with motor images (Broca's centre), but interfering with the connection of visual images (angular gyrus) with the same. This ischæmia would affect the connecting fibres in their passage beneath the Rolandic convolutions, thus causing the paresis of the inferior facial. The author compares the symptom called "dysantigraphia" with the dyslexia of Bruns, where the patient, after reading fluently a few words, finds it impossible to continue.

C. MACFIE CAMPBELL.

**A CONSTANT PALPEBRAL SYMPTOM IN PERIPHERAL FACIAL (125) PALSY.** DUPUY-DUTEMPS and R. CESTAN, *Journ. de Neurol.*, Feb. 5, 1904, p. 48.

WHEN a patient with peripheral facial palsy is asked to look down, the upper eyelid on the paralysed side follows the eye downward although it remains at an appreciably higher level than the upper



eyelid on the sound side. If now he is asked to close his eyes firmly, the upper eyelid on the paralysed side is *raised* abruptly, exposing the globe, and the eye is rotated upwards. The lower eyelid moves in a similar way, but to a less extent. The physiological explanation is simple, being based on the two facts, that the eyelids follow the movements of elevation and depression of the eyes because of the anatomical connections between the sheaths of the recti muscles and the eyelids, and that the eyes are normally turned upwards in energetic closure of the eyelids. The tendency for the upper eyelid to be raised in forcible closure of the eyes is physiological, but in the normal state it is almost completely masked by the antagonistic action of the orbicularis: it naturally becomes very marked where the orbicularis is paralysed, as is the case in peripheral facial palsy.

The authors have noted this phenomenon in fifteen consecutive cases of facial palsy and they therefore believe that it is always present, and that it constitutes a special sign of paralysis of the orbicularis, since it is absent in lagophthalmus, due to other causes, *e.g.* Basedow's disease, various forms of exophthalmus, cicatricial retraction of the eyelids, etc.—in all these the palpebral fissure is narrower and the upper eyelid is lower during forcible closure of the eyes than when the eye is simply made to look downward.

A. W. MACKINTOSH.

**OBJECTIVE SENSORY DISTURBANCES IN ACROPARÆSTHESIA  
(126) AND THEIR DISTRIBUTION ACCORDING TO NERVE-  
ROOTS.** J. DEJERINE and M. EGGER, *Rev. Neurol.*, Jan. 30,  
1904.

THE name Acroparæsthesia has been given to the presence of certain subjective sensations—prickling, tingling—in an extremity. Pick of Prague has called attention to the topographical distribution of these sensations in a case published by him, which corresponded to the distribution of spinal nerve-roots.

The authors confirm Pick's observation, but call attention to the presence in cases observed by them of permanent areas of hyperæsthesia corresponding to the distribution of nerve-roots. In two cases this anæsthesia occupied an area corresponding to the distribution of the 8th cervical and 1st dorsal nerves.

Considering the existence of these disturbances of objective sensibility and their root distribution, the authors conclude that acroparæsthesia is due to an irritative lesion of the posterior roots in their course within the cord.

C. MACFIE CAMPBELL.

**INVOLUNTARY MOVEMENTS OF THE HEAD AS EXPRESSION  
(127) OF AN ISOLATED AFFECTION OF ONE OF THE SEMI-  
CIRCULAR CANALS IN MAN.** OKOUNEFF, *Archiv. Internat.  
de Laryng.*, mars-avril 1904.

VERTIGO and nystagmus as indicative of labyrinthine lesions have been shown to have very considerable significance. Okonneff describes in detail two cases characterised, apart from vertigo, etc., by rhythmic movements of the head (in the first case), from behind forwards and from left to right, at a rate of 60 to 70 per minute; of the head and body (in the second case), at a rate of 110 to 120 per minute. Other indications pointed to disease of the labyrinth; and arguing from familiar physiological experiments on pigeons, the author diagnosed disease of *one* of the semi-circular canals.

Further clinical observations are wanted to throw light on this interesting and little known subject.

S. A. KINNIER WILSON.

**A PARTIAL CONTRACTION OF THE EXTENSOR COMMUNIS  
(128) DIGITORUM AS AN INDEX OF THE REFLEX EXCITA-  
BILITY OF THE UPPER LIMB (MIDDLE FINGER  
PHENOMENON).** E. SCIAMANNA, *Ann. dell' Istituto Psichiat. d.  
R. Università di Roma*, Vol. ii., 1902-1903.

THE author remarks that none of the arm reflexes that have as yet been investigated have proved to be of the same service in diagnosis as those that can be elicited in the lower limbs. He has devoted special attention to a more or less partial reaction exhibited by the *extensor communis digitorum*, which he finds to be of considerable diagnostic value.

If in a healthy adult the arm is made to rest in the semiflexed position, the palmar surface of the forearm being supported in such a way that the hand hangs loosely, and if the ulna is then tapped with a Wintrich hammer about two finger-breadths below the external condyle of the humerus, a movement of extension takes place in the middle finger independently of the other fingers. The movement thus elicited, Sciamanna proposes to call "the middle finger phenomenon."

The deviations of this reflex from the normal may be represented by means of the following five types of reaction: *A*, Absence of movement; *B*, Isolated movement upon relatively heavy percussion; *C*, Fairly isolated movement upon light percussion; this, together with movement of the hand, upon heavy

percussion; *D*, Accompanied by movement of the hand even upon light percussion; *E*, Accompanied also by movement of the fingers.

These types correspond to degrees of reflex excitability of the upper limb, which are further indicated by the greater or less rapidity of the elevation of the middle finger, the more or less complete extension of the phalanges, and the return of the finger in a more or less flaccid or tonic manner. The author has found that in the more marked cases of exaggerated tonicity and reflex hyper-excitability, the middle finger presents the reaction indicated by the types *D* and *E*. In cases in which there is diminished reflex excitability and flaccidity, the types *A* and *B* are found. He has also been able to observe that in not a few morbid conditions of the cervical and dorsal cord (cervical poliomyelitis, tabetic lesions of the cervico-dorsal region, etc.), and in many forms of peripheral neuritis affecting the upper limbs, the characters of the middle finger phenomenon serve to indicate a diminution of the reflex excitability that cannot be recognised in any other way. In cerebral and cerebro-spinal lesions, not attended by a distinct accentuation of the muscular tone and reflex movements as ascertained by other methods, he has been able to determine that this reaction generally has the type *C* or *D*. He claims that by the investigation of this phenomenon it is possible to recognise very slight degrees of increase or decrease in the reflex activity of the upper limb.

W. FORD ROBERTSON.

## PSYCHIATRY.

**CONTINUOUS ALCOHOLIC DELIRIUM.** SOUKHANOFF and (129) WEDENSKY, *Nouv. Icon. de la Salpêtrière*, No. 6, 1903, p. 391.

THE authors call particular attention to the well-marked distinctions existing between the form of mental disease which they call continuous alcoholic delirium and delirium tremens; also between continuous alcoholic delirium and chronic alcoholic dementia.

They consider that continuous alcoholic delirium differs from delirium tremens principally in the length of its duration. In some cases it persists for several years, and in no case is of brief duration. Further, after the first acute symptoms have subsided, the disease is marked by characteristic delusions, which do not occur in delirium tremens. Continuous alcoholic delirium is to be distinguished from chronic alcoholic dementia, we are told, by the absence of any marked alteration in the psychic personality of the

sufferer, and of a systematic development of delusions, both of which characteristics are to be observed in all cases of chronic alcoholic dementia. In the latter disease we find usually delusions of persecution referred to definite persons, accompanied by a suspicious and aggressive attitude. The sufferer from continuous alcoholic delirium is, on the contrary, good-humoured and sociable, and never, as we have said above, the victim of a systematised delusion. Moreover, chronic alcoholic dementia is a progressive disease, whereas continuous alcoholic delirium is scarcely ever so.

Alcoholic delirium is easily distinguishable from alcoholic melancholia. The emotional state in the latter disease is characterised by extreme depression and misery; while in alcoholic delirium there is no painful emotion, excepting what is caused by fear, and that is of short duration.

The diagnosis of continuous alcoholic delirium is somewhat difficult in the early stages of the disease. The malady is usually preceded by attacks of delirium tremens, and its morbid phenomena at first resemble those of delirium tremens. As soon as the acute symptoms subside, however, the nature of the complaint becomes apparent from the existence of hallucinations and illusions of hearing. The patient complains of being troubled by "voices"; he speaks of these "voices" critically, as of something outside his own personality, and, as a rule, converses quite rationally. The voices usually come to him from both right and left; occasionally it is an inner voice which he hears. The sound values of the voices vary according to the gravity of the case. In slight cases the voices are neither continuous nor very loud. The patient is able to attend to business in spite of them, and may appear quite rational. In acute cases, the voices are so loud and numerous that the patient is unable to occupy himself with anything else; he is always listening to the voices, and sometimes they even set up a condition of mental confusion. It is found that the voices are always more imperative after fatigue than after rest.

The content of the auditory hallucinations in continuous alcoholic delirium is fairly uniform. For example, the patient hears himself being reproached for having led a depraved life, or for having abused strong drinks. He is sometimes being insulted, and often hears obscene language. Other voices praise and encourage him. Not infrequently the voices urge him to curse God, hinder him from prayer, and so on.

The attitude of consciousness towards these voices is generally one of criticism; the patient recognises them as an abnormal phenomenon—an illness, but very often, especially if he belongs to the uneducated classes, tends to think that they come from the devil.

Beside the hallucinations of hearing, which are one of the

fundamental manifestations of continuous alcoholic delirium, there may also be hallucinations of smell, sight and touch. One patient of the authors' complained that "they"—that is to say, his invisible interlocutors—"poisoned the air with bad smells."

Continuous alcoholic delirium may last for years without resulting in any marked enfeeblement of the sufferers' intellectual capacity. On the contrary, the tendency is for the morbid phenomena to weaken gradually, and finally disappear.

Lesions of the peripheral auditory apparatus very often exist in combination with continuous alcoholic delirium. They are generally unilateral, and result in gradual impairment of hearing. There is, of course, no immediate connection between the two diseases. Strong hallucinations of hearing exist in cases where there is no lesion.

One circumstance of the disease most favourable to recovery is the fact that the patient usually leaves off drinking. This may be due to the fear that if he persist in drinking he will lose his reason altogether. There is another theory advanced by Prof. Korsakoff to account for this fact, namely, that the craving for strong drink ceases with the onset of the disease. With regard to the connection between continuous alcoholic delirium and other alcoholic psychoses, the authors prefer not to commit themselves to any definite conclusions for the present; they hazard the theory, however, that where continuous alcoholic delirium follows delirium tremens, as it most frequently does, it may be considered, not as developing out of delirium tremens, but as an accessory complication of the fundamental malady. The authors point out that there is a great deal of literature in all the principal Continental languages on the subject of the disease of which their paper treats, but that little exact information concerning it is given. The most detailed and precise account of the complaint is found in the Manual of Psychiatry of Prof. Korsakoff. This author is of opinion that continuous alcoholic delirium is the second stage of delirium tremens, and that it sets in after the critical sleep.

The statistics of the psychiatric hospital in Moscow give some interesting facts concerning the disease.

Out of 4813 patients under treatment in the hospital between 1887 and 1903, thirty-three were suffering from continuous alcoholic delirium. We see, therefore, that this is a comparatively rare disease. Thirty of the sufferers were men; three only women.

Hereditary predisposition was shown in 96.55 per 100 of these cases. These figures show sufficiently the enormous significance of heredity in the etiology of continuous alcoholic delirium. In this respect, the proportion showing predisposition

is higher than in the case of any other form of mental disorder.

In taking statistics of age, we find that the malady occurs most frequently between the ages of 26 and 35.

Most of its victims belong to the classes whose associations are of an unintellectual nature, *e.g.* farmers, factory hands, artisans, etc.

F. LEECH.

**THE CLINICAL FORMS OF CHRONIC ALCOHOLISM.** HANS (130) EVENSEN. Lecture at the Psychical Clinic, Gaustad Asylum.

DR EVENSEN states that in Norway the consumption of alcohol is very small, not exceeding  $1\frac{1}{2}$  litres of absolute alcohol per individual per annum. As factors in the production of insanity, psychical causes and bodily illnesses are much more important. The figures as to causation for the ten years 1886-1895 are given as follows:—

	THE TWO SEXES TOGETHER.		MEN ONLY.	
	Contributive Cause per cent.	Sole Cause per cent.	Contributive Cause per cent.	Sole Cause per Cent.
Hereditary Disposition	48·1	31·9	45·6	33·3
Psychical Causes	28·8	18·9	23·3	15·5
Bodily Diseases	19·7	14·4	20·9	15·4
Alcoholic Excess	7·7	5·4	14·5	10·1

The author reviews briefly the symptoms of the different clinical varieties of alcoholic disturbance. The relationship between degeneration and alcoholism is considered, and the conclusion arrived at is that alcoholism is not necessarily a manifestation of inherited degeneration, and that the alcoholic psychoses often appear in individuals in whom hereditary predisposition cannot be shown. Thus, in the ten years, 1886-1895, alcohol was the cause of mental disorder in 22 per cent. of men without demonstrable hereditary predisposition, and in only 5·4 per cent. was there heredity.

As regards epilepsy and alcohol, the author says it has not yet been proved that drink can be a cause of epilepsy in a person not predisposed to the latter disorder. When, however, one considers how frequently convulsions occur in alcoholics (Kræpelin, "typical epilepsy" in one-third of such; v. Krafft-Ebing, in 10 per cent.), one cannot avoid the conclusion that alcohol is capable of producing various epileptiform seizures, though epilepsy proper may

not be induced, just as paralytic dementia or cerebral tumour may do.

The author discusses, with illustrative cases, the instances of psychoses with alcohol as the cause, which occurred for a period of six years prior to the date of his lecture at the Asylum at Gaustad. These fall into the following groups: (1) depressive forms, (2) expansive, (3) aments, (4) dementa, (5) paranoiacs, (6) pseudo-paralytica. In reference to treatment of the alcoholic subject, it is pointed out that the latter's weak will and amenability to suggestion would give scope for "psychical therapy." Attempts to wean him from his habit are not as a rule attended by lasting results in private practice, and under these circumstances it is probably best to place the patient on a progressively smaller daily dose of alcohol. Immediate deprivation of alcohol should, the author thinks, be the plan adopted in institutional treatment, and is not then attended by any great risk. Treatment should last not less than one year. At present, the author regrets to state, there is deficiency of asylums for alcoholic subjects in Norway.

EDWIN GOODALL.

**THE GENESIS OF CERTAIN SYMPTOMS IN CATATONIC (131) STATES.** W. ALTER, *Neurol. Centralbl.*, 1904, p. 8.

In this paper Alter endeavours to give an explanation of certain symptoms of what he calls negativism, which are met with in some cases, exhibiting catatonic phenomena. In some he recognises these symptoms as being secondary in character, that is, they are due to hallucinations or delusions. In some, however, they are primary, and it is these he seeks to account for. Every movement brings into play two sets of muscles, the protagonist and the antagonist. The former are the muscles acting in the direction of the desired movement, the latter act in the reverse direction and control and co-ordinate the former. If for any reason the path of the nervous impulses to the former becomes blocked, or is rendered abnormally difficult, the nervous energy flows along the other channels most usually associated with these, that is, to the antagonist muscles, and as a result, a movement, the exact negative of that expected, is brought about, and to an exaggerated degree. He believes there is good ground for thinking that such a condition of things actually occurs. Structures which, in the normal state, may exhibit a condition corresponding to cathelectronus, may, when pathologically affected, exhibit a state resembling anelectronus. Injured vitality, alterations in metabolism or in the chemical or molecular structure of the nervous elements concerned, are the pathological

conditions which he thinks may bring about the change. He cites Berger's investigations of the blood in catatonia and Löwenfeld's of the urine in hysteria, as proofs that such conditions exist.

In these manifestations of negativism it is probable that the motor apparatus is concerned; but in cases where the manifestations of disorder assume the form of automatism or reiteration, it is probable that the sensory apparatus is affected. There is a dissociation of the normal congruence of the volition of a movement and the sensory representation of its accomplishment. Where the latter fails, the former may persist, and thus catalepsy, stereotypy, or reiterative movements may result. The same causes which produce negativism may also produce the above mentioned dissociation, but in the former the defect is in the centrifugal paths, while in the latter it is in the centripetal. There are other minor suggestions in the paper, but they also are purely speculative, and would be very difficult to demonstrate and consequently to refute. That is to say, they are equally difficult of proof or of disproof.

JAS. MIDDLEMASS.

**CLINICAL AND CRITICAL CONTRIBUTION TO THE STUDY  
(132) OF CATATONIA. PATINI and MEDIA, *Ann. di Patol.*, Anno  
xxi., F. v. and vi.**

THE subject of catatonia, about which so much has been written recently, receives, in this paper, a thorough investigation. The authors give details of ten cases, which they consider to be typical of this condition.

These cases may be divided into three classes—(a) six which showed a complex of hallucinatory symptoms followed by stupor; (b) one epileptic in whom stupor supervened; and (c) two who had previously shown signs of melancholia atonita. In the early stages some were in the condition of exaltation, others were depressed, and others were confused, but they all passed into the stuporose condition, and *flexibilitas cerea* appeared soon after.

It is this last symptom which the authors consider to be the essential element of the catatonic state, and to it alone they suggest that the term "catatonia" should be applied. Besides these symptoms, stereotypy of language, of movement, and of pose were frequent; negativism was also seen. They do not admit, however, that the above complex of symptoms deserves to be considered a distinct clinical entity, but they regard the catatonic condition as an episodic symptomatic state, which is intimately connected with stupor, and often intercurrent with it in various mental diseases.



The authors then review the opinions of Kahlbaum and Kraepelin, who maintain that catatonia should be considered a separate clinical entity, and also those of Seglas, Chaslin, and many others who oppose this position.

Kahlbaum bases his opinion on two orders of facts—the cyclic course of the disease, and the presence of so-called characteristic symptoms, amongst which he includes exaltation, the habit of declaiming aloud with much histrionic display, stereotypy of phrase and of movement, “*flexibilitas cerea*,” and negativism.

But the authors maintain that these symptoms indicate a condition of confusion and disorientation, and that they cannot be considered as characteristic of any one disease. Frequently they owe their origin to a hallucinatory condition, which Kahlbaum and Kraepelin noted, but they attached little importance to it.

They then proceed to examine the above symptoms more fully and critically, and they pay special attention to the condition of *flexibilitas cerea*.

This condition, following in the wake of the stupor, is generally slightly marked at first, and it gradually becomes more intense as time goes on, i.e. that in the early stage, the unusual position into which the limb of the patient is placed, is maintained for a short time only, sometimes just while the operator is present; but later the time is increased, and may reach fifteen to twenty-five minutes in the upper limb, but not so long in the lower limb. In the earlier stages also the patient may be made by frequent repetition of an order to carry out a small movement; later all spontaneous movement is lost. They also note that in most early cases if the limb is subjected to rapid and varied movements, it may pass either into a condition of absolute rigidity, or the muscles may become completely relaxed.

When the condition of *flexibilitas cerea* has become pronounced, the patient is often still sensitive to pain, the cutaneous reflexes are brisk, the patellar reflex is increased, the Achilles-jerk is normal, vascular reflexes are excessive, the pupils react, electrical stimulation of muscles gives normal reactions, and the temperature is subnormal. The muscles of the face are never affected.

The above symptoms of the catatonic state may be divided into two groups—(a) those which are manifestations of psychic activity—stereotypy of phrase and of movement; and (b) those which are the result of psychic anergia—*flexibilitas cerea* and negativism.

Those of the first group are considered to be the result of a narrowing down of the active psychic sphere, which renders possible the existence of one isolated representation, which remains undisturbed by any other concurrent and antagonistic motor representations (in the sense of James), or the persistence

of one physiological process in a field of consciousness relatively empty (in the sense of Müller).

In offering an explanation of the condition of *flexibilitas cerea*, the authors refer to the comparison of Bianchi between this condition and induced hypnotism; and they suggest that while in the condition of hypnosis the sensory areas still functionate, although in a disordered manner, in this *flexibilitas cerea* only one sensory area is still functioning, and that is the kinæsthetic-motor area. Through this alone can suggestions be made to the patient. This is done when we place a limb in an unusual position, and this position is maintained because no antagonistic stimuli arrive to disturb it, and because the "residuum of the former activity" (Müller) renders it easy for the muscular action to be prolonged. In negativism, they suggest that the catatonic state supervenes in a patient whose former mental derangement showed a strong hostility to the outer world, and this hostile condition impresses itself on the catatonic state.

Next attention is given to the differences between the catatonic and the cataleptic states. Of these differences, besides the diversity of origin, of duration, of course, and of exit, they mention:—

1. In catalepsy the muscular plasticity may be extended or generalised and the muscles of the face may be affected; in catatonia this is not possible.

2. In cataleptics closure of the eyes causes the patient to pass into the lethargic condition; in catatonia no change takes place.

3. In catalepsy sensibility and reflexes are abolished; in catatonia they are not.

Referring to the relation between catatonia and dementia præcox, the authors suggest that the former occurs as a symptom in the course of the latter. They admit that both conditions arise in the same degenerate soil, and that both appear about the same period of organic development, but they do not admit that they are the same thing.

Moreover, while dementia præcox has only one ending, that of dementia, a recovery from the catatonic condition is possible, especially in the early stages.

R. G. Rows.

**THE PATHOGENESIS OF THE SPECIFIC DELUSIONS IN  
(133) GENERAL PARALYTICS.** ADAM WIZEL, *Neurolog. Centralbl.*,  
July 16 and Aug. 1, 1903.

THE delusions of general paralytics possess very characteristic features which distinguish them from those of other mental diseases, showing customarily the wildest extravagance, the most

extraordinary hyperbolism. The question thus arises as to what is the psychological mechanism determining this condition, and in some part to answer this Dr Wizel has carried out an experimental investigation of the appreciation of time and space relations in general paralytics as compared with that in normal individuals and in secondary dementa.

The method of examination was simple. With regard to appreciation of time, the patients were ordered to attend to the beating of a metronome for definite periods of time and then asked to state roughly the length of time occupied. To prevent counting the beats the metronome was set at its highest speed (200 per minute). Only such cases were investigated in whom a recognition of ordinary time divisions still obtained and all outside sources of possible distraction were excluded.

The times chosen for each seance were  $\frac{1}{2}$ , 2, 1,  $\frac{1}{4}$ ,  $1\frac{1}{2}$  and 3 minutes. The following abstract from Dr Wizel's numerous tables will give some idea of the marked failure of time appreciation in general paralytics as compared with normal individuals and ordinary dementa.

	DURATION OF EXPERIMENT.					
	$\frac{1}{2}$	2	1	$\frac{1}{4}$	$1\frac{1}{2}$	3
Average of . . . .						
Normal . . . . .	0.66	3.08	1.41	.52	2.86	4.1
Dementa . . . . .	1.06	3.73	2.54	0.83	2.71	5.27
General Paralytics . .	3.5	7.5	23.75	0.15	50	50

This failure Dr Wizel found as markedly in space as in time appreciation. Asked to state in yards the length of a wall of the room in which they were, to guess how many people it could contain, and to arrange beans according to simple patterns before them, the results given by general paralytics were surprisingly inferior to those of ordinary dementa. In the general paralytic, then, says Dr Wizel, time and space presentations are entirely or almost entirely obliterated, on account of which delusions bearing upon time and space relations encounter no obstacle in judgment and attain to grotesque exaggeration and unbounded hyperbolism.

The author has here confined his investigations within narrow bounds, but states his conviction that in general paralytics the objective determination of appearances and things, movement, force, weight, that is all of the properties of matter, is defective; the defect depending on the partial or complete obliteration of many presentations.

R. CUNYNGHAM BROWN.

**TREATMENT.****ON THE TREATMENT OF AFFECTIONS OF THE EAR AND  
(134) IN PARTICULAR OF AURICULAR VERTIGO BY LUMBAR  
PUNCTURE.** BABINSKI, *Ann. des Mal. de l'Oreille et du Larynx*,  
fév. 1904.

THE author has already shown (*Soc. Méd. des Hôp.*, 7 Nov. 1902. and 24 April 1903) that the application of the continuous current to the mastoid processes produces nausea, vertigo, nystagmus and a lateral movement of the head towards the positive pole. He further found that many diseases of the ear diminish or even counteract entirely this electric vertigo (*Soc. de Biol.*, 26 Jan. 1901 and 25 April 1903); and again, that in healthy individuals, the removal of a certain quantity of cerebro-spinal fluid by lumbar puncture usually diminishes the resistance to this same artificial giddiness.

From this series of observations one may construct a syllogism leading logically to the therapeutic value of lumbar puncture in certain ear diseases.

The results given in the paper demonstrate conclusively the great, sometimes almost marvellous, effect which abstraction of 15 to 20 c.c. of cerebro-spinal fluid may have on deafness, auditory vertigo, and humming or other noises in the ears. One of Babinski's patients, aged 35, a deaf mute from the age of 2½ years, after three successive lumbar punctures, is now able to hear the voice at a distance of 20 centimetres.

S. A. KINNIER WILSON.

**SURGICAL TREATMENT OF FACIAL PARALYSIS.** MUNCH,  
(135) *Sem. Méd.*, mars 9, 1904.

AN interesting critical account is given of the various recorded cases of surgical intervention in facial paralysis. The general conclusion is come to that the operation, while of course theoretically admissible, is of little value practically, a good facial result being obtained usually at the expense of some other part. From this point of view, hypo-glosso-facial grafting is preferable to spino-facial. References to English, German and American work on the subject.

S. A. KINNIER WILSON.

**ON STRETCHING OF THE NERVES IN RAYNAUD'S DISEASE.**  
(136) DE BOVIS, *Sem. Méd.*, fév. 17, 1904.

IN advanced cases of this disease, even those which have reached a gangrenous stage, the author has obtained excellent and enduring results (as far as the vasomotor and trophic alterations are con-

cerned) by practising elongation of the nerve trunks interested (ulnar, median, radial). Details of two cases, considered by the author as cured. At one of the operations he found indications of peripheral neuritis. He holds that the term Raynaud's disease must be restricted to symmetrical gangrene absolutely independent of any pre-existing pathological affection.

S. A. KINNIER WILSON.

**PUERPERAL ECLAMPSIA TREATED BY LARGE DOSES OF  
(137) THYROID EXTRACT.** H. OLIPHANT NICHOLSON, *Journ. of  
Obstet. and Gynaecol. of the Brit. Empire*, Jan. 1904, p. 32.

IN previous papers the administration of some preparation of the thyroid gland had been suggested. (1) As a prophylactic remedy in the pre-eclamptic state, small doses of from 5 to 20 grains in the day being recommended. Several cases in this stage of the disease had been treated most successfully. (2) When actual convulsions were present. Then very large doses were necessary—30 or 40 grains, soon repeated if required, the object being to produce symptoms of thyroid intoxication as rapidly as possible. The essential thing in treatment at this stage was to get the renal function established. In eclampsia there is a marked constriction of the smaller blood-vessels, including those of the kidneys, with a resulting rise in blood pressure; consequently *vaso-dilatation* is the principle of treatment indicated, and this is the only way in which the kidneys can be made to secrete again. Thyroid extract is an ideal vaso-dilator in such cases, but as had been shown in a previous communication, a full dose of morphine hypodermically acts promptly and powerfully in a similar manner. The combination of these two remedies seemed to provide a very successful method of treating many cases of eclampsia, and the secretion of urine became very rapidly re-established under their use. The following case illustrated this.

Mrs A., aged 21 years, primipara, showed marked œdema of the abdominal walls and of the body generally on July 20th, though the urine contained no albumen. Pulse was abnormally slow, and the sphygmogram was characteristic of a greatly increased peripheral resistance. On August 4th, patient was drowsy, complained of headache and dimness of vision with vomiting. Next day labour commenced, anuria developed, and an eclamptic fit occurred in the evening. She became quite comatose, and a living child was born at 7 P.M. A second fit followed the expulsion of the placenta. Half a grain of morphine was injected. A third but somewhat less severe fit occurred an hour after delivery. At 11 P.M. 40 grains of thyroid extract were given

in the form of tabloids. A catheter showed that there were only 3 drachms of urine in the bladder, and on boiling it became solid. Next morning (Aug. 6th) patient was fairly conscious, and the bladder was greatly distended; 40 ounces of urine were drawn off by the catheter; 30 grains of thyroid were then given, and the drowsiness gradually passed off. In the evening symptoms of "thyroidism" were evident, but a third dose of thyroid (15 grains) was given. The patient had taken 85 grains in twenty-three hours, with the result that the re-establishment of the renal function was unusually rapid and well maintained. In less than twelve hours 40 ounces of urine were secreted. Thyroid was continued during the first ten days of the puerperium.

AUTHOR'S ABSTRACT.

---

## Review

**ARBEITEN AUS DEM NEUROLOGISCHEN INSTITUTE AN DER WIENER UNIVERSITÄT.** Herausgegeben von Prof. OBERSTEINER. Heft. ix., 1903, pp. 427, 6 plates, 97 figures. Deuticke, Vienna. Price 25s.

IF the aim of a review of a scientific work be to bring it to the notice of those interested in its subject, one of the above titled book, the latest volume of Professor Obersteiner's excellent series, seems highly desirable, and its excellence is such that it can scarcely remain a thankless task.

It contains the result of work by Obersteiner himself, and by his assistants and pupils, on various nervous subjects, anatomical, human and comparative, pathological, and clinical and pathological.

As most of the papers have already been abstracted for this journal, little mention is needed of the contents of the volume.

Obersteiner contributes an interesting paper on the variation in position of the pyramidal tracts; Tarasewitsch deals with the finer anatomy of the basal ganglia and the fibre-tracts in connection with them; Karplus writes on the Australian brain and that of other low types; Hatschek gives the result of investigation of some of the tegmental tracts of the mid-brain; and Kreuzfuchs his estimation of the surface area of the cerebellum.

Comparative anatomy is represented by papers by Hatschek, Schlesinger, Zuckerkandl and Schacherl, all of considerable interest and worth.

But the pages, and they form the greater bulk of the volume,

which contain both clinical and pathological descriptions of carefully investigated cases, will be the most welcome to the majority of neurologists, for the everywhere apparent accuracy of observation and the careful correlation of symptoms with their anatomical basis could not fail to be instructive even though each worker brought to light no new facts or offered no new explanation of something hitherto observed.

In this way Gerber and Matzenauer deal with the similarity of leprosy and syringomyelia; Frankl-Hochwart describes a case of oculo-motor palsy of neuritic nature; while the papers of Halban and Infeld on the symptomatology of mid-brain lesions and by Breuer and Marburg on apoplecticiform bulbar palsy deserve special notice.

An admirable feature of each paper is the full consideration the literature on the subject it treats of receives, and the full citation of earlier authorities cannot fail to be of great service to all who avail themselves of the service of the volume.

The volume is well printed and its figures and illustrations are clear and excellent.

GORDON HOLMES.

## Bibliography

### ANATOMY

KLEIST. Experimentell-anatomische Untersuchungen über die Beziehungen der hinteren Rückenmarkswurzeln zu den Spinal-ganglien. *Virchow's Arch.*, März 1904, S. 381.

GOLDSTEIN. Versuch einer Einteilung der Rückenmarkshinterstränge. *Deutsche Ztschr. f. Nervenheilk.*, Bd. xxv., H. 5-6, 1904, S. 456.

LAQUEUR. Noch einmal die Lage des Centrums der Macula lutea im menschlichen Gehirn. *Virchow's Arch.*, März 1904, S. 407.

GIGLIOLI. Nuovi studi sul cervello. *Riv. crit. di clin. Med.*, Feb. 13, 1904, p. 107.

PROBST. Das kindliche Gehirn. Reuther & Reichard, Berlin, 1904, M. 4.

MIES and BARTELS. Über die grösste Breite des menschlichen Hirnschädels. *Ztschr. f. Morphol. u. Anthropol.*, Bd. vii., H. 1, 1904, S. 63.

BARTELS. Untersuchungen und Experimente an 15,000 menschlichen Schädeln. *Ztschr. f. Morphol. u. Anthropol.*, Bd. vii., H. 1, 1904, S. 81.

KLIEN. Ueber die Konfiguration der lumbalen Intervertebralräume. *Mitt. a. d. Grenz. der Med. u. Chir.*, Bd. xii., H. 5, 1904, S. 635.

### PHYSIOLOGY

DÉJÉRINE. Quelques considérations sur la théorie du neurone. *Rev. Neurol.*, mars 15, 1904, p. 205.

BORDONI. Contro la teoria del neurone. *Clin. moderna*, Feb. 24, 1904, p. 88.

LANGLEY and ANDERSON. On the Union of the Fifth Cervical Nerve with the Superior Cervical Ganglion. *Journ. Physiol.*, Feb. 25, 1904, p. 489.

- CARLSON. The Rate of the Nervous Impulse in the Spinal Cord and in the Vagus and Hypoglossal Nerves of the California Hagfish. *Am. Journ. Physiol.*, March 1904, p. 401.
- ZWAARDEMAKER und QUIX. Ueber die Empfindlichkeit des menschlichen Ohres für Töne verschiedener Höhe. *Arch. f. Anat. u. Physiol.*, H. 1 u. 2, 1904, S. 25.
- TATSUSABURO SARAI. Untersuchungen über die Lage der Bogengänge des Labyrinthes im Schadel und über die Flüssigkeitsverschiebung in den Bogengängen bei Kopfbewegungen. *Ztschr. f. Ohrenheilk.*, Bd. xlii., H. 1-2, 1903, S. 72.
- KURT GOLDSTEIN. Kritische und experimentelle Beiträge zur Frage nach dem Einfluss des Zentralnervensystems auf die embryonale Entwicklung und die Regeneration. *Arch. f. Entwicklungsmechanik d. Organ.*, Bd. xviii., H. 1, 1904, S. 57.
- HARTLE. Ueber den Einfluss von Wasser und anistotonischen Kochsalzlosungen auf die Grundfunktionen der quergestreiften Muskelsubstanz und der motorischen Nerven. *Arch. f. Anat. u. Physiol.*, H. 1 u. 2, 1904.
- FLETCHER. The Osmotic Properties of Muscle, and their Modifications in Fatigue and Rigor. *Journ. Physiol.*, Feb. 25, 1904, p. 414.
- BRODIE and DIXON. Contributions to the Physiology of the Lungs. On the Innervation of the Pulmonary Blood Vessels; and some Observations on the Action of Suprarenal Extract. *Journ. Physiol.*, Feb. 25, 1904, p. 476.
- J. BEARD. A Morphological Continuity of Germ-Cells as the Basis of Heredity and Variation (concluded). *Rev. of Neurol. and Psychiat.*, March 1904, p. 185.
- WENIG. Über neue Sinnesorgane der Isopoden. *Rivnác, Prag*, 1904, M.—40.
- BELMONDO. Curva miografica e "curva ergografica." *Riv. di Patolog. nerv. e ment.*, Vol. ix., f. 2, 1904, p. 81.
- GIULIO OBICI. Azione del Radium sulla eccitabilità della corteccia cerebrale. *Riv. di Patolog. nerv. e ment.*, Vol. ix., f. 2, 1904, p. 76.

## PSYCHOLOGY

- FISCHER. Die ästhetischen Anschauungen Gottfrieds Sempers und die moderne psychologische Ästhetik. *Arch. f. d. ges. Psychol.*, Bd. xi., H. 4, 1904, S. 362.
- WUNDT. Über empirische und metaphysische Psychologie. *Arch. f. d. ges. Psychol.*, Bd. xi., H. 4, 1904, S. 333.
- LARGUIER des BANCELS. De la Mémoire. *Arch. de Psychol.*, fév. 1904, p. 145.
- KOZLOWSKI. Le plein et le vide. *Arch. de Psychol.*, fév. 1904, p. 178.
- GOETHUYSEN. Das Mitgefühl. *Ztschr. f. Psychol. u. Physiol. d. Sinnesorgane*, Bd. xxxiv., H. 3 u. 4, 1904, S. 161.
- BONNIER. La perception de trépidation. *Rev. Neurol.*, mars 15, 1904, p. 219.
- GEORGES VERNET. De l'orthophrénopédie. *Ann. méd.-psychol.*, mars-avril 1904, p. 227.
- LEMAITRE. Audition colorée hallucinatoire, stabilité et hérédité des photismes. *Arch. de Psychol.*, fév. 1904, p. 164.
- KÖHLER. Der simultane Farben- und Helligkeitskontrast mit besonderer Berücksichtigung des sogenannten Florkontrastes. *Arch. f. d. ges. Psychol.*, Bd. xi., H. 4, 1904, S. 423.

## PATHOLOGY

- DANA. On Cytodiagnosis in Nervous Diseases. With a Description of the Technique of Cell-diagnosis. *F. W. Hastings, Med. Rec.*, Jan. 23, 1904.
- FRAENKEL. Lymphocytosis of the Cerebro-spinal Fluid. *Med. Rec.*, Jan. 23, 1904.
- WARRINGTON. Note on the Ultimate Fate of Ventral Cornual Cells after Section of a Number of Posterior Roots. *Journ. Physiol.*, Feb. 25, 1904, p. 503.
- PFISTER. Zur Kenntnis der Mikropie und der degenerativen Zustände des Centralnervensystems. *Neurol. Centralbl.*, März 16, 1904, S. 242.
- MINGAZZINI. Experimentelle und pathologisch-anatomische Untersuchungen über den Verlauf einiger Bahnen des Centralnervensystems (Fort.). *Monatsschr. f. Psychiat. u. Neurol.*, Bd. xv., H. 3, 1904, S. 223.
- NAGEOTTE. Contribution à l'étude anatomique des cordons postérieurs. (Un cas de lésion de la queue de cheval et un cas de tabes incipiens.) *Nouv. Icon. de la Salpêtrière*, jan.-fév. 1904, p. 17.



- JENDRASSIK. Das Prinzip der Bewegungseinrichtung des Organismus. (Beiträge zur allgemeinen und speziellen Pathologie.) *Deutsche Ztschr. f. Nervenheilk.*, Bd. xxv., H. 5-6, 1904, S. 347.
- ARMAND-DELILLE. Rôle des poisons du bacille de Koch dans la méningite tuberculeuse et la tuberculose des centres nerveux. Steinheil, Paris, 4 fr. 50.
- BALLET. Les rayons N en pathologie nerveuse. *Journ. des Praticiens*, mars 19, 1904, p. 181.
- BALLET. Les rayons N. Leur recherche dans quelques affections du système nerveux. *Presse méd.*, mars 16, 1904, p. 169.
- TIDAUX. Le diagnostic entre les fibromes utérins, les affections nerveuses et la grossesse. *Journ. des Praticiens*, mars 19, 1904, p. 183.

## CLINICAL NEUROLOGY AND PSYCHIATRY

### GENERAL—

- JOHN MILSON RHODES. On the Alleged Increase of Diseases of the Nervous System. *Brit. Med. Journ.*, March 12, 1904, p. 598.

### MUSCLES—

- V. BECHTEREW. Ueber den Muskelwulst bei Myotonie. *Neurol. Centralbl.*, März 16, 1904, S. 255.
- VALENTIN. Über den klonischen Krampf des Musculus tensor veli und die dadurch erzeugten objektivhörbaren Ohrgeräusche. *Ztschr. f. Ohrenheilk.*, Bd. xvi., H. 1-2, 1904, S. 84.

### PERIPHERAL NERVES—

- BERNHARDT. Ueber einige seltener vorkommende periphere Lähmungen. *Berl. klin. Wchnschr.*, März 7, 1904, S. 237.
- W. E. TAYLOR. Un cas de névrite du plexus brachial suivi d'autopsie. *Rev. Neurol.*, fév. 29, 1904, p. 153.
- BREGMAN. Ueber eine traumatische Lähmung des Ramus volaris profundus n. ulnaris. *Neurol. Centralbl.*, März 16, 1904, S. 261.
- TENNYSON PATMORE. Deep Ulnar Nerve Paralysis and Functional Powers. *Brit. Med. Journ.*, March 12, 1904, p. 596.
- C. H. CATTLE. Bilateral Neuritis of Fifth and Sixth Cervical Nerves. *Brit. Med. Journ.*, March 12, 1904, p. 596.

### SPINAL CORD—

- Tabes.**—ERNEST S. REYNOLDS. Some Unusual Cases of Tabes Dorsalis with Remarks. *Rev. of Neurol. and Psychiat.*, March 1904, p. 177.
- PAPADOPOULOS. Contribution à l'étude de l'ataxie locomotrice progressive. *Prog. méd.*, mars 5, 1904, p. 145.
- RAYMOND. Crises tabétiques en ceinture. *Journ. des Praticiens*, fév. 27, 1904, p. 135.
- Combined Sclerosis.**—CROUZON. Des scléroses combinées de la moelle. *Thèse de Paris*, fév. 25, 1904.
- CROUZON. Anatomie pathologique des scléroses combinées tabétiques. *Nouv. Icon. de la Salpêtrière*, jan.-fév. 1904, p. 52.
- Primary Lateral Sclerosis.**—LANGDON. Primary Lateral Sclerosis. *Cincinnati Lancet Clinic*, Jan. 30, 1904.
- Multiple Sclerosis.**—BARTELS. Zur Frage der Regeneration der Nervenfasern in den Herden der multiplen Sklerose. *Neurol. Centralbl.*, März 1, 1904, S. 195.
- Spine.**—OHRAN ABDI. Ueber einen Fall von chronischer Arthritis ankylopoetica der Wirbelsäule. Fraktur der Wirbelsäule und Quetschung der Cauda equina. *Mitt. a. den Hamburg. Staatskrankenanstalten*, Bd. iv., H. 2, 1904, S. 57.

SCHANZ. Die statischen Belastungsdeformitäten der Wirbelsäule mit besonderen Berücksichten der kindlichen Skoliose. Enke, Stuttgart, 1904, M. 9.

ALFRED GORDON. Contribution to the Study of Spondylose Rhizomyélique, with report of Three Cases. *Med. Rec.*, Feb. 13, 1904, p. 247.

#### BRAIN AND MEMBRANES—

**Meningitis.**—RIST et PARIS. Contribution à l'étude clinique et expérimentale de la méningite cérébro-spinale à diplocoques de Weichselbaum. *Arch. gén. de méd.*, fév. 23, 1904, p. 449.

EMIL ADLER. Ein Fall von spontaner transitorischer Glykosurie bei Meningitis cerebrospinalis. *Ztschr. f. Heilk.*, Bd. xxv., H. 11, 1904, S. 1.

PORTER PARKINSON. A Case of Post-basal Meningitis due to Pneumococcus Lanceolatus: Recovery. *Brit. Journ. of Child. Dis.*, Vol. i., No. 3, 1904, p. 112.

FAURE et LAIGNEL-LAVASTINE. À propos des lésions des méningites cérébrales aiguës. *Arch. gén. de méd.*, mars 15, 1904, p. 641.

**Thrombosis of Cerebral Sinus.**—STENGER. Die otitische Hirnsinusthrombose nach den in der Ohrenklinik der Charité gesammelten Beobachtungen. Hartung, Königsberg, 1904, M.—80.

**Perencephaly.**—SOMMER. Porenkephalie und cerebrale Kinderlähmung sur psychopathologischen Nomenklatur. *Monatschr. f. Psychiat. u. Neurol.*, Bd. xv., H. 3, 1904, S. 219.

**Microcephalus.**—A. de BLASIO. Un microtatocephalo fra i microcefali. *Riv. mens. de psychiat. forense*, Vol. vii., Num. 1, 1904, p. 19.

**Hemiplegia.**—HENRY FRASER. A Case of Hemiplegia occurring during Whooping-cough and Diphtheria. *Brit. Med. Journ.*, March 12, 1904, p. 597.

**Intracranial Tumours.**—FRANK R. FRY. A Cerebellar Tumour: Operation; Recovery. *Journ. Nerv. and Ment. Dis.*, March 1904, p. 157.

THEODORE DILLER. A Brain Tumour involving the Superior Parietal Convolution. *Am. Journ. Med. Sc.*, March 1904.

DIXON MANN. Cerebral Tumour accompanied by Psychical Symptoms. *Med. Chron.*, March 1904, p. 376.

RAYMOND. Tumeur cérébrale avec abolition des réflexes tendineux. *Hérissay, Evreux*, 1904.

**Cerebral Abscess.**—KÖLPIN. Zur Symptomatologie und pathologischen Anatomie des Hirnabscesses. *Deutsche Ztschr. f. Nervenheilk.*, Bd. xxv., H. 5-6, 1904, S. 465.

**General Paralysis.**—SAVAGE. Two Lectures on General Paralysis of the Insane. *Clin. Journ.*, March 9, 1904, p. 321.

CULLERRE. Un nouveau cas de paralysie générale conjugale. *Hérissay, Evreux*, 1904.

KERAVAL et DANJEAN. L'état du fond de l'œil chez les paralytiques généraux. *Arch. de Neurol.*, mars 1904, p. 193.

CH. FÉRÉ. Note sur l'aplatissement hypotonique du pied chez les paralytiques généraux. *Nouv. Icon. de la Salpêtrière*, jan.-fév. 1904, p. 79.

BOUCHAUD. Kystes séreux du pavillon de l'oreille et othématome dans la paralysie générale. *Ann. méd.-psychol.*, mars-avril 1904, p. 204.

**Injury to Skull.**—FEDERSCHMIDT. Ein Beitrag zur Kasuistik der Schädelverletzungen. *München. med. Wchnschr.*, März 15, 1904, S. 483.

#### GENERAL AND FUNCTIONAL DISEASES—

CORNELIUS. Die Druck- oder Schmerzpunkte als Entstehungsursache der sogenannten funktionellen Nervenerkrankungen, ihre Entstehung und Behandlung. Perles, Wien, 1904, M. 1.

**Chorea.**—FRANK. Zur Kenntnisse der Chorea chronica progressiva. *Wien. klin. Wchnschr.*, März 3, 1904, S. 247.

**Paralysis Agitans.**—T. STUART HART. Paralysis Agitans; Some Clinical Observations based on the Study of 219 Cases seen at the Clinic of Professor M. Allen Starr. *Journ. Nerv. and Ment. Dis.*, March 1904, p. 177.

F. RAYMOND. La maladie de Parkinson. *Nouv. Icon. de la Salpêtrière*, jan.-fév. 1904, p. 1.

- FRIEDLAENDER. Bewegungstherapie bei Paralysis agitans. *Ztsch. f. d. d. u. physik. Therapie*, Bd. vii., H. 1-2, 1904, S. 635.
- Epilepsy.—M. ALLEN STARR. Is Epilepsy a Functional Disease? *Journ. Nerv. and Ment. Dis.*, March 1904, p. 145.
- ONUF. Some Features of the Epileptic Attack. *New York State Journ. of Med.*, Feb. 1904, p. 71.
- PARIS. Epilepsie: pathogénie et indications thérapeutiques. *Arch. de neurol.*, fév. et mars 1904, pp. 99 et 206.
- MORIZ MANN. Beiträge zur Lehre über die Rindenepilepsie. *Wien. klin. Wchnschr.*, März 3, 1904, S. 252.
- MERKLEN et HEITZ. Exagération du pouvoir toxique du bromure sous l'influence de la déchloruration dans un cas d'épilepsie. *Soc. méd. des hôp.*, fév. 18, 1904, p. 134.
- ENRIQUEZ et GRENET. Hyperchloruration et épilepsie. *Soc. méd. des hôp.*, fév. 11, 1904, p. 100.
- TOULOUSE. Minéralisation et hypochloruration dans le traitement bromuré de l'épilepsie. *Soc. méd. des hôp.*, fév. 4, 1904, p. 79.
- Hysteria.—Thiemich. Hysterie im Kindesalter. Bergmann, Wiesbaden, 1904.
- VORSTER. Ueber hysterische Dämmerzustände und das Vorbeireden. *Monatschr. f. Psychiat. u. Neurol.*, Bd. xv., H. 3, 1904, S. 161.
- NONNE. Nachtrag zu meinem Aufsatz "Ueber zwei durch zeitweiliges Fehlen der Patellar-reflexe ausgezeichnete Fälle von Hysterie." *Deutsche Ztsch. f. Nervenheilk.*, Bd. xxv., H. 5-6, 1904, S. 487.
- LANNOIS et POROT. Sur les hémispasmes de la face; hémispasme facial vrai, hémispasme hystérique. *Lyon méd.*, fév. 7, 1904, p. 234.
- BÉCLÈRE. Un cas de mutisme hystérique guéri par suggestion au cours de l'examen radioscopique. *Soc. méd. des hôp.*, fév. 4, 1904, p. 81.
- WOLLENBERG. Die Hypochondrie. *Pathologie und Therapie* (Nothnagel), Bd. xii., 1904, M. 1.40.
- Neuralgia.—LEDERER. Neuralgia due to Dental Irritation. *Med. Rec.*, March 5, 1904, p. 372.
- Tetanus.—TOURNEAU. Drei Fälle von Tetanus. *Deutsche med. Wchnschr.*, März 3, 1904, S. 347.
- WALLACE AND SARGENT. Four Cases of Tetanus treated by Injections of Antitoxin into the Spinal Theca. *Lancet*, March 5, 1904, p. 642.
- Tetany.—BECK. Ein Beitrag zur Tetanie im Kindesalter. *Jahrb. f. Kinderheilk.*, März 1904, S. 277.
- H. HYSLOP-THOMSON. Tetany associated with Muco-membranous Colitis and Phthisis. *Brit. Med. Journ.*, March 12, 1904, p. 597.
- FREUND. Tetanie als Ursache der Starrbildung. *Wien. med. Wchnschr.*, März 5, 1904, S. 406.
- Writers' Cramp.—GUSTAF NORSTROM. A Study of the Affection, "Writers' Cramp." *N. Y. Med. Journ.*, March 12, 1904, p. 491.
- BORCHARDT. Der Schreibkrampf und die ihm verwandten Bewegungsstörungen, ihre Entstehung, Bedeutung und zweckmässige Behandlung. Steinitz, Berlin, 1904, M. 2.
- Exophthalmic Goitre.—KLIEN. Ueber die Bedeutung der bei Morbus Basedowii im Zentralnervensystem nachgewiesenen pathologisch-anatomischen Befunde. *Deutsche Ztsch. f. Nervenheilk.*, Bd. xxv., H. 5-6, 1904, S. 431.
- Trophoneuroses.—PARKES WEBER. A Case of Erythromelalgia, illustrating its Relation to Raynaud's Symptom-complex: "Diffusion" of the Phenomena during Period of Exacerbation. *Brit. Journ. of Dermatol.*, Feb. 1904.
- DIEHL. Ueber die Raynaud'sche Krankheit. *Centralbl. f. Nerven- u. Psychiat.*, Feb. 15, 1904, S. 99.

#### MENTAL DISEASES—

- KRAEPELIN. Psychiatrie. Ein Lehrbuch für Studierende und Ärzte. Barth, Leipzig, 1904, M. 23.
- NISSL. Kritische Bemerkungen zu Ziehen's Aufsatz: "Ueber einige Lücken

- und Schwierigkeiten der Gruppierung der Geisteskrankheiten." *Centralbl. f. Nervenhk. u. Psychiat.*, März 15, 1904, S. 171.
- HELLBRONNER. Ueber die Aufgaben der klinischen Psychiatrie. Kemink & Zoon, Utrecht, 1904, M.—60.
- MÖNKEMÖLLER. Tortar und Geisteskrankheit. *Allg. Ztschr. f. Psychiat.*, Bd. lxi., H. 1 u. 2, 1904, S. 58.
- J. SHAW. The Physiognomy of Mental Diseases and Degeneracy. Simpkin, London, 1904, 8/4.
- SHUTTLEWORTH. On Mental Deficiency in Children. *Brit. Journ. of Children's Dis.*, Vol. i., No. 3, 1904, p. 99.
- WIDOWITZ. Ueber die geistige Ermüdung der Schulkinder. *Wien. klin. Wchnschr.*, März 10 u. 17, 1904, S. 277.
- PICK. Über einige bedeutsame Psycho-Neurosen des Kindesalters. Marhold, Halle, 1904, M.—80.
- SCHULZE. Über moral insanity. Ein Beitrag zur Psychologie des moralischen Irreseins. *Allg. Ztschr. f. Psychiat.*, Bd. lxi., H. 1 u. 2, 1904, S. 109.
- DARCANNE. Contribution à l'étude clinique des monologues chez les aliénés. Hérissay, Evreux, 1904.
- JUNG. Über manische Verstimmung. *Allg. Ztschr. f. Psychiat.*, Bd. lxi., H. 1 u. 2, 1904, S. 15.
- MADELEINE PELLETIER.—L'association des idées dans la manie aiguë et dans la débilité mentale. Rousset, Paris, 1904.
- W. H. B. STODDART. Motor Symptoms of Mania and Melancholia. *Lancet*, March 5, 1904, p. 639.
- PFERSDORFF. Ueber intestinale Wahnideen im manisch-depressiven Irresein. *Centralbl. f. Nervenhk. u. Psychiat.*, März 15, 1904, S. 161.
- MOURATOFF. Contribution à l'étude de la démence catatonique. Hérissay, Evreux, 1904.
- MARANDON DE MONTYEL. Obsessions et Impulsions. *Arch. d'Anthropol. crim.*, T. xix., No. 122, 1904, p. 81.
- SELVATICO ESTENSE. Donne uomini. *Riv. mens. de psichiat. forense*, Vol. vii., Num. 1, 1904, p. 1.
- LADAME. La rage expérimentale à virus fixe et ses lésions histologiques. *Journ. de Neurol.*, fév. 20 et mars 5, 1904, p. 61 et 81.
- GANHÖR. Ein kasuistischer Beitrag zur Imbecillität. *Allg. Ztschr. f. Psychiat.*, Bd. lxi., H. 1-2, 1904, S. 1.
- KOENIG. Die Ätiologie des einfachen Idiotie verglichen mit derjenigen der cerebralen Kinderlähmungen. *Allg. Ztschr. f. Psychiat.*, Bd. lxi., H. 1 u. 2, 1904, S. 133.
- W. B. PRITCHARD. Delirium Grave: A Critical Study, with Report of a Case with Autopsy. *Journ. Nerv. and Ment. Dis.*, March 1904, p. 162.
- JOSÉ INGEGNIEROS. Simulation de la folie, précédé par une étude sur la simulation dans la lutte pour la vie. *Arch. d'Anthropol. crim.*, T. xix., No. 122, 1904, p. 137.
- V. CRABE. Ein Fall von episodischer Katatonie bei Paranoia. *Allg. Ztschr. f. Psychiat.*, Bd. lxi., H. 1 u. 2, 1904, S. 40.
- GIGLIOLI. Del torcicolle mentale. *Riv. crit. di clin. Med.*, Feb. 13 e 20, 1904, pp. 104-121.
- CAUDMONT. Le Torticollis mental. État mental du tiqueur. Morel, Lille, 1904.
- YANNIRIS. L'aliénation mentale en Grèce. Étude statistique. *Ann. méd.-psychol.*, Jan. 1904, p. 60.
- Dementia Præcox.**—PRITCHARD. Observations on Dementia Præcox. *Cleveland Med. Journ.*, Jan. 1904.
- MASSELEN. La démence précoce. Joanin, Paris, 1904.
- WLADIMIR SERBSKY. Contribution à l'étude de la démence précoce (suite et fin). *Ann. méd.-psychol.*, mars-avril 1904, p. 188.
- Criminal Psychiatry.**—A. DE BLASIO. Urantropo sanguinario. *Riv. mens. de psichiat. forense*, Vol. vii., Num. 1, 1904, p. 12.
- Forensic.**—BATTY TUKE and HOWDEN. The Relations of Insanities to

Criminal Responsibility and Civil Capacity. *Edin. Med. Journ.*, March 1904, p. 244.

Treatment.—J. CHRISTIAN. Un médecin d'asile accusé d'avoir fait mourir de faim un de ses malades. *Ann. méd.-psychol.*, mars-avril 1904, p. 219.

SCHULTZE. Über Psychosen bei Militärfangenen nebst Reformvorschlägen. Fischer, Jena, 1904, M. 6.

LUDWIG. Über die Anlage besonderer Speiseküle in den öffentlichen Irrenanstalten. *Allg. Zeitschr. f. Psychiat.*, Bd. lxi., H. 1 u. 2, 1904, S. 169.

#### ALCOHOL, ETC.—

BERTILLON. L'Alcoolisme et les Moyens de le combattre jugés par l'expérience. Firmin-Didot et Cie, Paris, 1904, 2 fr.

LIVINGSTON. Ergot in Alcoholism, Morphinism, and the General Class of Drug Habit Cases. *Med. News*, March 5, 1904, p. 445.

ERNBERG. Über Intoxication mit per os eingenommenem Chloroform. *Nordiskt Med. Arkiv*, Feb. 22, 1904.

SAMUEL BELL. Some Toxemias in Relation to the Production of Nervous and Mental Diseases. *Journ. Am. Med. Assoc.*, Feb. 20, 1904, p. 507.

#### CRANIAL NERVES—

WITTMACK. Die toxische Neuritis acustica und die Beteiligung der gehörigen Ganglien. *Zeitschr. f. Ohrenheilk.*, Bd. xvi., H. 1-2, 1904, S. 1.

CAPGRAS. Relations des maladies unilatérales de l'oreille avec les hallucinations de l'ouïe. Hérissey, Evreux, 1904.

WILSON PARRY. A Case of Paroxysmal Labyrinthine Vertigo associated with Special Ocular Symptoms and alleviated by Seton. *Lancet*, March 5, 1904, p. 649.

TATSUSABURO SARAI. Herpes der Ohrmuskel mit Neuritis des Nervus facialis. *Zeitschr. f. Ohrenheilk.*, Bd. xvi., H. 1-2, 1904, S. 186.

PAINBLAN. Un cas de paralysie variolique du moteur oculaire externe. *Echo méd. du Nord*, fév. 14, 1904, p. 78.

Pupil.—BUMKE. Beiträge zur Kenntnis der Irisbewegungen. *Centralbl. f. Nervenh. u. Psychiat.*, Feb. 15, 1904, S. 89.

REICHARDT. Ueber Pupillarfasern im Sehnerv und über reflektorische Pupillenstarre. *Deutsche Zeitschr. f. Nervenheilk.*, Bd. xxv., H. 5-6, 1904, S. 408.

ABELSDORFF und NAGEL. Ueber die Wahrnehmung der Blutbewegung in den Netzhautkapillaren. *Zeitschr. f. Psychol. u. Physiol. d. Sinnesorgane*, Bd. xxxiv., H. 3 u. 4, 1904, S. 291.

NAGEL und SCHAEFER. Ueber das Verhalten der Netzhautzapfen bei Dunkeladaptation des Auges. *Zeitschr. f. Psychol. u. Physiol. d. Sinnesorgane*, Bd. xxxiv., H. 3 u. 4, 1904, S. 271.

NAGEL. Einige Beobachtungen über die Wirkung des Druckes und des galvanischen Stromes auf das dunkeladaptierte Auge. *Zeitschr. f. Psychol. u. Physiol. d. Sinnesorgane*, Bd. xxxiv., H. 3 u. 4, 1904, S. 285.

BACH. Was wissen wir über Pupillenreflexentren und Pupillenreflexbahnen? Karger, Berlin, 1904, M. 2.

Facial Paralysis.—V. SARBÓ. Zur Pathogenese der sog. rheumatischen Facialislähmung. *Deutsche Zeitschr. f. Nervenheilk.*, Bd. xxv., H. 5, 6, 1904, S. 398.

VITEK. Ein Beitrag zum primären tonischen Gesichtskrampf mit Muskelwogen. *Neurol. Centralbl.*, März 16, 1904, S. 257.

GROLHOUS. Le spasme facial, étude clinique et diagnostique. *Thèse de Paris*, fév. 1904.

#### REFLEXES—

KURT MENDEL. Ein Reflex am Fusrücken. *Neurol. Centralbl.*, März 1, 1904, S. 197.

VERGER et ABADIE. Étude graphique des réflexes plantaires à l'état normal et dans quelques affections spasmodiques du système pyramidal. *Nouv. Icon. de la Salpêtrière*, jan.-fév. 1904, p. 67.

REH. Ueber das Verhalten der Reflexe bei Hirntumoren. *Monatschr. f. Psychiat. u. Neurol.*, Bd. xv., H. 3, 1904, S. 182.

## MISCELLANEOUS SYMPTOMS—

- ZILLIER. Contribution à l'étude des troubles nerveux dus à la présence des végétations adénoïdes. *Thèse de Paris*, fév. 24, 1904.
- C. BIRT. Lesion of Post-central Convolution without Paralysis. *Brit. Med. Journ.*, March 12, 1904, p. 598.
- SPITZMÜLLER. Ein Fall von Neurofibromatose. *Wien. med. Wchnschr.*, Feb. 27, 1904, S. 366.
- MIBELLI. A proposito di alcuni casi di neurodermite cronica lineare. *Sperimentale*, Feb. 1904, p. 29.
- MARINESCO. Sur deux cas de paralysie flasque dus à la compression du faisceau pyramidal sans dégénérescence de ce dernier, avec signe de Babinski et absence des réflexes tendineux et cutanés. *Rev. Neurol.*, mars 15, 1904, p. 210.
- TATSUSABURO SARAI. Ein Fall von akustisch-optischer Synästhesis (Farbenhören). *Ztschr. f. Ohrenheilk.*, Bd. xlvi, H. 1-2, 1904, S. 180.
- ALEXANDER MORISON. The Neural Factor in Heart Disease. *Edin. Med. Journ.*, March 1904, p. 235.
- REHFISCH. Nervöse und kardiale Arrhythmie. *Deutsche med. Wchnschr.*, März 10 u. 17, 1904, S. 332.
- MINOR. Ueber die Localisation und klinische Bedeutung der sogen. "Knochen-sensibilität" oder des Vibrationsgefühls. (Schluss.) *Neurol. Centralbl.*, März 1, 1904, S. 199.
- NORSTRÖM. La migraine; traitement par le massage. *Baillière et fils*, Paris, 1904, 2 fr. 50.
- ELIOT GORTON. Insomnia. *Med. Rec.*, Feb. 20, 1904, p. 298.
- PEUGNIEZ et PHILIPPE. Hémisection traumatique de la moelle (syndrome de Brown-Sequard). *Hérissay*, Evreux, 1904.
- WESTPHAL. Ueber Bewegungserscheinungen an gelähmten Augenmuskeln in einem Fall von Korsakowchen Psychose. *Berl. klin. Wchnschr.*, Feb. 22, 1904, S. 183.
- Speech.**—APT. Zwei Fälle von Störungen der Sprache in Folge von Zwangsvorstellungen. Koenig, Leipzig, 1904, M. 1.
- ALTER. Ein Fall von Sprachstörung. *Monatschr. f. Psychiat. u. Neurol.*, Bd. xv, H. 3, 1904, S. 214.
- BRETON. L'aphasie urémique. *Journ. des Praticiens*, fév. 27, 1904, p. 131.
- MEHNERT. Über Sprachstörungen mit besond. Berücksicht. des Stammels und Stotterns bei Schulkindern. Urban, Dresden, 1904, M.—75.
- G. PERUSINI. Un caso di alalia idiopatica di Coën complicata a probabile stenosi mitralica. Audimutismo ed alalia idiopatica di Coën. *Riv. di Patolog. nerv. e ment.*, Vol. ix, f. 2, 1904, p. 49.

## TREATMENT—

- ADRIANO VALENTI. Aromatici e nervini nell'alimentazione. Hoepli, Milano, 1904.
- DÉJÉRINE. Le traitement des hystériques à l'hôpital. *Bull. méd.*, fév. 24, 1904, p. 169.
- G. BALLEZ. De la nécessité de traiter, et de traiter d'une façon active et précoce, les hystériques, dans les services hospitaliers. *Soc. méd. des hôp.*, fév. 18, 1904, p. 155.
- ABRAHAM. Ueber Versuche mit "Veronal" bei Erregungszuständen der Paralytiker. *Centralbl. f. Nervenheilk. u. Psychiat.*, März 15, 1904, S. 176.
- H. HIGIER. Zur Therapie der Neurasthenia sexualis (Heroinum muriaticum). *Neurol. Centralbl.*, März 18, 1904, S. 256.
- DARIER. Note on the Action of Radium as an Analgesic and Nerve Tonic. *Lancet*, March 5, 1904, p. 652.
- V. BECHTEREW. Heilgymnastische Behandlung im Bade. *Centralbl. f. Nervenheilk. u. Psychiat.*, März 15, 1904, S. 180.
- Surgical.**—BOCHENHEIMER. Der N. facialis in Beziehung zur Chirurgie. *Arch. f. klin. Chir.*, Bd. 72, H. 8, 1904, S. 461.
- TAYLOR and PIERCE CLARK. The Surgical Treatment of Facial Palsy, with the Technique of Facio-hypoglossal Nerve Anastomosis. *Med. Rec.*, Feb. 27, 1904, p. 321.
- Electrical.**—RÖMER. Ueber die Wirkung des galvanischen Stroms auf Tetanus-Gift, Tetanus-Antitoxin und Toxin-Antitoxin-Gemische. *Berl. klin. Wchnschr.*, Feb. 29, 1904, S. 209.

# Review of Neurology and Psychiatry

---

## Original Articles

### THE DIAGNOSTIC VALUE OF THE PLANTAR REFLEX.

By STANLEY BARNES, M.D., B.Sc., M.R.C.P. (Lond.), M.B.,  
Ch.B. (Birm.),

Pathologist and Medical Registrar to the Queen's Hospital, Birmingham;  
late House Physician to the National Hospital for the Paralysed and  
Epileptic, London.

It was in 1896 that the abnormal type of plantar reflex was first described by Babinski as occurring in cases of disease or injury of the pyramidal tracts. Since that year a large number of observations have been recorded by various authors, but as yet there is no unanimity as to the diagnostic value of the reflex. The statement of Babinski that in adults an abnormal plantar reflex ("Babinski sign" or "extensor response") indicates organic disease has been the essential contention. This statement has been upheld by a large number of writers, notably by Collier, Van Gehuchten, Brissaud, Boeri, Buzzard, Kalischer, Walton and Paul, Langdon, Van Epps, and Fraenkel and Collins; whilst on the other hand the value of the reflex has been called in question by Schüler, Giudiciandrea, Judson Bury, Cohn, Harris and others.

It is unfortunate that in many of the communications on this subject more care has not been taken to describe the method adopted in testing the reflex; it might then be possible to harmonise to a far greater extent than at present is the case the results of all investigators.

During my two years' residence as house-physician at the National Hospital for the Paralysed and Epileptic, London, I was

enabled to test this reflex—often repeatedly—under the best possible conditions. I have also had the opportunity during the last twelve months of testing the reflex in the patients at the Queen's Hospital, Birmingham. In all, therefore, this paper is based on the examination of about 2500 patients; of this number, about fifty per cent. were suffering from organic nervous disease, about twenty per cent. from "functional" nervous conditions, and the rest from general diseases unassociated with nervous symptoms. In about 150 cases, post-mortem evidence was obtained as to the correctness of the diagnosis.

My deepest thanks are due to Dr Hughlings Jackson, Sir William Gowers, Dr Ferrier, Dr Ormerod, Dr Turner, Sir Victor Horsley and Mr Ballance for permission to refer to cases which occurred at Queen Square; and to Dr Foxwell and Dr Kauffmann for allowing me to use cases of theirs from the Queen's Hospital, Birmingham.

#### CLINICAL EXAMINATION.

In almost all cases the patients were in bed, and there can be no doubt that it is more convenient to put the patient in the horizontal position whenever there is any difficulty in obtaining the reflex otherwise. It is essential that the patient's attention should be distracted as fully as possible from the real object in view, so that no volitional movement shall interfere with those which are purely reflex. It is thus advisable that he should be unable to see either the hand of the physician or his own foot at the time of stimulation. In very few cases did I then find any difficulty in distracting the patient's attention sufficiently to cause complete relaxation of the muscles of the lower extremities. I am quite certain that in any borderland case, at any rate, it is useless to attempt to obtain the reflex unless the toe, ankle and knee-joints are quite supple.

It sometimes happens that with the patient fully extended in bed in the supine position complete relaxation cannot be obtained; in these cases I adopt the position recommended by Collier (*Brain*, 1898, p. 73), the knee being bent and resting on a pillow, and the hip flexed and rotated outwards. But I do not think that as a rule this position is necessary or gives the most delicate results (see later).



The stimulus should always be a minimal one; that is to say, the smallest stimulus which will provoke a definite movement of the metatarso-phalangeal joint of the big toe. If a stronger stimulus is applied the movement of the toe may be masked in the coarser movements of the foot and leg, or may be disturbed by the rapid occurrence of voluntary movements. I have found drawing the thumb or finger nail along the sole to be as a rule the most convenient means of eliciting the reflex; it is easy thus to increase the stimulus by pressing more firmly and moving the nail more rapidly over the sole; if a still stronger stimulus is required, a quill or pencil-point may be used. The stroke should be from heel to toe.

The foot must always be warm and dry, so that the sole shall be sensitive and its natural friction shall be preserved.

I found that the most constant and delicate results were produced by stimulation of the outer part of the sole, that is, the edge of the sole corresponding roughly to the outer and lower surfaces of the fifth metatarsal and adjacent tarsal bones. The sole here is not quite so sensitive as at the inner side, so that frequently a stronger stimulus is necessary to evoke the reflex from the outer side of the sole than from the inner side; but in all cases when patients are "ticklish," it is far easier to obtain from this position a constant result with a minimum of spreading of the movements so as to involve the foot and leg. Apart from this reason is the fact that I have repeatedly obtained an "extensor response" (Babinski sign) by stimulation of the outer side of sole when a (normal) flexor response resulted if the inner side were irritated (see Case 13).

Nothing seems to be known as to the rationale of the movements of the other toes. The rule is that the big toe is extended and the small ones are spread out (abducted) and slightly extended in all cases of severe pyramidal disease, especially in young adults; whilst in health there is usually flexion of the five digits together. But in some cases, with flexion of the hallux is combined extension of the other digits; more frequently the big toe is extended and the smaller ones are flexed. It seems clear that in doubtful cases the movements of the smaller toes are untrustworthy; and the other movements of foot, leg and thigh do not appear to have much practical value. For this reason it has recently been customary in testing this

reflex to confine attention almost solely to the movements of the big toe, *i.e.* to determine whether flexion or extension occurs at the metatarso-phalangeal joint. In some "borderland" cases, a movement in one direction, quickly followed by one in the other, is observed; the *first* movements should then be taken as determining the nature of the reflex.

I think it well to adopt Collier's nomenclature and refer to the plantar reflex as being of either of the "flexor type" or of the "extensor type," the latter being synonymous with "Babinski's sign." Most of us are agreed that it is always better, where a particular physical sign can be shortly and adequately expressed in physical language, to refer to it constantly in that way, rather than to tack on to it the name of the discoverer of the sign; we prefer the term "absent knee-jerk" to "Westphal's sign," and we retain the term "Argyll Robertson pupil," chiefly because there is no short phrase to express adequately that combination of conditions. I shall therefore throughout refer to plantar stimulation resulting in a "flexor response," or an "extensor response," as the case may be.

#### CONDITIONS UNDER WHICH A FLEXOR RESPONSE OCCURS.

This is the normal plantar reflex in man. It occurs only when the pyramidal tracts have reached a certain stage of development, so that children up to the age of twelve months usually do not show it. The flexor response may be normally absent up to the age of four, though it is rare to find a healthy child above the age of two years in whom the reflex is extensor. After this age the character of the reflex persists (apart from disease) to extreme old age.

The flexor response is often present on the diseased side in cases of hemiplegia of long duration. In my experience it has never been present in a hemiplegia of less than several years' duration, and nearly always in cases in which some contracture has occurred. In hemiplegic patients in whom the extensor response is, so to speak, dying out, one can often obtain a flexor response by stimulating the inner surface of the sole with the knee strongly flexed; whereas, if the outer side of the sole is stimulated and the knee and hip are kept extended, an extensor response may be obtained.

The flexor response is also often present in cases of functional paralysis of the lower extremity, although in the severer cases the plantar reflex is almost invariably absent. Certain cases of neurasthenia and hysteria show very brisk knee-jerks, and often give an ankle clonus which is very difficult to differentiate from what occurs in organic cerebro-spinal disease; in such cases the plantar reflex is, if present at all, invariably flexor.

Some cases of definite pyramidal tract disease in an early stage give a flexor response, and this fact has often been used as an argument against its value as a clinical sign. Considering, however, that in a severe pure hemiplegia a certain amount of degeneration always occurs in the crossed pyramidal tract of the other ("unaffected") side, and that in these cases it is extremely rare to get an extensor response, although increased knee and ankle jerks are the rule, I do not think we should be surprised that in slight affections of the pyramidal tracts a flexor response may occur. I shall refer to this subject again later.

#### WHEN DOES THE EXTENSOR RESPONSE OCCUR?

An extensor response occurs in all cases of recent pyramidal disease of more than slight severity, provided that a plantar reflex is present at all.

It is a well-known fact that in central apoplexy and allied conditions the knee-jerk becomes exaggerated often only several days after the lesion has occurred, and it has been assumed by some that this increase is dependent upon secondary degeneration occurring in the spinal cord. However this may be, it is certain that the plantar reflex depends upon no such factor; Marinesco (*Archiv. de Neurologie*, June 1903) observes that in hemiplegia the Babinski reflex appears shortly after the "stroke." I would go further and say that *the extensor response comes on simultaneously with the lesion of the pyramidal system*. I have several times noted cases in which an operation for central tumour involved injury to the Rolandic cortex; as soon as the patient began to come round from the anæsthetic, usually before consciousness had fully returned, the plantar reflex, which had been flexor before the operation, was now of the extensor type; it always remained extensor after the effects of the anæsthetic had passed off (see Case 1).

The extensor response does not occur in all cases of pyramidal disease. If the disease is of long standing, a flexor response may be present. If the disease is only slight—if presumably an insufficient number of pyramidal fibres is destroyed—the reflex may remain of flexor type. Probably if about one-tenth of the pyramidal fibres passing to the lumbosacral enlargement are *suddenly* thrown out of action by organic disease an extensor response will occur; whereas if a smaller number are affected, the response will remain flexor. On the other hand, if a slow progressive degeneration, from whatever cause, occur in the pyramidal tracts, it seems as if as many as one-half of the fibres of the pyramidal tracts may be thrown out of function without necessarily causing an extensor response. (See Case 13.)

The extensor response also occurs under certain other conditions. It is occasionally seen in cases of *myopathy*; it is undoubtedly rare to find it in this disease, and when present it seems more probable that it indicates the presence of lateral sclerosis of the cord (such as has been described in this disease) than that it indicates a fallacy in the general rule that the extensor response only occurs in pyramidal disease.

In cases of *tabes* the movements are often confusing, especially if stimulation of the sole is painful, or the patient unusually "ticklish," but apart from other disease of the spinal cord or brain—disease involving the pyramidal tract—I have never seen in *tabes* a typical extensor response.

By far the most important of the conditions under which an extensor response occurs, apart from organic affections of the pyramidal system, is *epilepsy*. It is the rule after an epileptic seizure that the plantar reflexes are of the extensor type for a time, which depends upon the severity of the attack and the recuperative power of the patient. I have never seen it occur in *petit mal*. After a fit of ordinary severity the plantar reflexes are extensor for from three minutes to an hour; in status epilepticus the reflex is in my experience invariably extensor throughout the attack, and may so remain for several days after the cessation of fits. If the convulsions are unilateral, the plantar reflex of the afflicted side is extensor, whilst that of the sound side is flexor. In the convulsions of children who have reached a stage of development at which the response is normally

flexor, it usually happens that the response becomes extensor for a time. In each of these diseases, whilst the patient is still unconscious after the fit, the reflex can usually be obtained, and is extensor in type. If the patient is artificially roused, by faradism or douching, it seems that the reflex much more rapidly becomes normal than when they are left to sleep off the effects of the fit.

On the other hand, I have never seen an extensor response occur after a *hysterical seizure*, however violent it might be. The reflex in these patients may be diminished or absent, but it is never changed in type; nor does any change seem to occur in cases of physical exhaustion from hard physical exertion.

In several cases of *uræmic convulsions* I have recently had the opportunity of observing, the reflex was changed from the flexor to the extensor type during and immediately after the fits.

In *meningitis* the plantar reflex often becomes extensor in type; this I have observed in meningitis due to the tubercle bacillus, to the pneumococcus and to the diplococcus intracellularis: in one case, I thought the infection might be influenzal (Case 10). From the few observations I have made in these conditions, the extensor reflex seems usually to be due to a rise in the intracranial pressure, rather than to direct pyramidal implication, and it only occurs when the patients are approaching a comatose state; but up to the present my observations are too few to speak with any certainty on the point.

In cases of *cerebral tumour* in which the pyramidal system was not involved (as proved post-mortem), I have often seen an extensor response, but never apart from signs of increased intracranial tension. As in meningitis, it seems that the same intracranial pressure which so affects the convolutions as to cloud the intellect or cause coma, can, by throwing out of gear the pyramidal system, cause a motor paresis or paralysis and a concurrent extensor plantar reflex. The same is also true of acute or chronic hydrocephalus (Case 4).

I have carefully observed many cases of toxic conditions—septicæmia, pyæmia, malignant endocarditis, pneumococcal and other forms of pneumonia, enteric fever, etc. In none of them was an extensor response present in the absence of meningitis, although symptoms of “meningism” were frequently present;

so that it seems clear that, apart from convulsive states and alterations in intracranial pressure, the pyramidal system cannot be so affected by the toxins of these organisms as to alter the character of the plantar reflex, although they may reduce the normal reaction. The extensor response has been recorded in cases of tetanus, of strychnine poisoning, and of opium poisoning, but I have had no opportunity of verifying these statements.

Excepting in children I have never observed any alteration in type of the plantar reflex in sleep or under anæsthetics. The reflex may be diminished or disappear, but it never changed from flexor to extensor, or *vice versa*.

I have had many opportunities of observing the plantar reflex in cases of rheumatoid arthritis, and I have thrice seen an extensor response occur in these cases. In each case the response was a sharp upward jerk of the hallux, such as is usually associated with slight pyramidal disease. In each case all the tendon-jerks were greatly exaggerated. I am unwilling to allow these cases as exceptions to the general rule, for in all three cases there was considerable fixation of the spinal column, and it is possible that the spinal cord was involved in an exudate from the inflamed joints. I know of no post-mortem evidence either proving or disproving this point.

In *severe anæmias* it is not uncommon to find an extensor response on both sides, associated with diminished or absent knee-jerks. As a rule, this condition only occurs in the late stages of such cases, when any nervous symptoms are masked by the general failure in health of the patient, and when the asthenia is so profound as to prevent any slight symmetrical loss of power from being observed. One such case, dying after hæmorrhage from various mucous membranes, has recently occurred at the Queen's Hospital (see Case 8). In this case it was found on staining sections of the spinal cord by the Marchi method that considerable degeneration was present in the pyramidal tracts, although during life no sign or symptom, other than the reflexes, suggested organic disease of the nervous system.

#### INFANTILE RESPONSE.

In children, plantar stimulation results in a sharp extensor movement of the hallux, with spreading (abduction) of the other

toes. This response is present in the new-born child, and usually persists up to two years of age. Occasionally, especially in children who are late in learning to walk, this "infantile response," as it has been aptly termed by Collier, is still present at the age of four or even five. Beyond this age its presence indicates a pathological condition, *i.e.* a condition of either acquired disease or delayed development. A healthy child up to the age of eight may give an infantile response if tested when soundly sleeping; an adult when asleep never in health gives an infantile or an extensor response.

The infantile response closely resembles that which occurs in the slighter forms of pyramidal disease in the adult, and that which is the result of plantar stimulation in the monkey (Collier), in which animal the pyramidal tracts are much less well-developed than in man. The fact that the plantar reflex of a new-born child is of this character, and not of the form seen in the severe spastic states of the adult, suggests that even at birth the pyramidal system is by no means functionless, although myelination of its fibres has hardly commenced.

#### ABSENCE OF THE PLANTAR REFLEX.

The plantar reflex is absent when the continuity of the reflex arc is destroyed, as by multiple neuritis, section of the internal popliteal or sciatic nerves, or pressure upon the sacral plexus; by poliomyelitis anterior in the fifth lumbar and first sacral segments; by pressure of tumours or caries upon this part of the cord.

It is often absent in cases of severe hysteria.

It was present in the one case of complete section of the cord above the lumbar region which I have seen (see Case 2).

It is absent also if the foot is cold or very moist. In very severe nerve "shock," and immediately before death, it disappears, but considerably later than does the knee-jerk.

It will be seen then that, with the exception of epilepsy and allied states, all the cases in which the extensor response occurs are those in which there is almost certainly organic disease of the pyramidal system. But should epilepsy be regarded as an exception? The post-epileptic state is one of intense exhaustion. The patient lies about in any position, too anæsthetic to feel

pain, too senseless to think, too flaccid to perform more than the smallest and most vital movements. He is, in fact, for the time being paralysed mentally and physically. In those cases in which Jacksonian attacks "of idiopathic origin" affect only one limb, it often happens that recovery of complete power after an attack is a matter of days or weeks. What wonder, then, that an exhaustion so severe as to produce a paralysis indistinguishable from that of organic disease should produce also an organic change in the character of the plantar reflex? Whether the cortical cells or the cortical fibres bear the brunt of the attack, it seems clear that for a time no impulses are transmitted through the pyramidal system. It is usual to regard idiopathic epilepsy as an organic disease; in this sense, the extensor response occurring in the immediate post-epileptic state is also indicative of organic disease of the pyramidal system.

#### TRANSITIONAL FORMS OF PLANTAR REFLEX—"PYRAMIDAL EQUILIBRIUM."

We have seen that the normal response is flexion of all the toes. In the well-marked abnormal or Babinski reflex, the hallux is deliberately extended at the metatarso-phalangeal joint, and the smaller toes are spread out or abducted from one another and slightly extended. If an otherwise healthy patient, who has just had a severe epileptic convulsion, is carefully watched, it will be found that the plantar reflex does not suddenly change from the typical extensor to the typical flexor type, but that an intermediate stage occurs. As the organic exhaustion wears away, the small toes cease to spread, and concurrently the extension of the big toe becomes quicker but of smaller range. As recovery continues, a state is soon reached at which it seems impossible to say whether the response is flexor or extensor. The stimulus necessary to provoke movement at the metatarso-phalangeal joint of the big toe is greater than before; and it is usually found that such a stimulus, applied to the outer edge of the sole with the limb extended, produces an extensor response; whereas if the inner side of the sole is stimulated, and especially if the knee and hip are flexed, a flexor response ensues. This, then, is a state in which a balance seems to be struck between the flexor and the extensor states; to continue the simile, by



alteration of position of the patient and the stimulus, we can to within certain limits change the position of the fulcrum of the balance, and cause at will a preponderance of the flexor or the extensor elements. Of this state it will be convenient to speak as the condition of *pyramidal equilibrium*. This condition is seen in conditions other than the post-epileptic state; and it is certain that its presence has been the cause of many mistakes in the past. It may occur in any pyramidal lesion when more than a certain portion of the pyramidal influence is removed, either by the cutting of some pyramidal fibres or by the action of pressure upon cells or tracts of fibres in reducing the conducting power of all these fibres. We have no accurate means of judging the severity of the compressing agent, but we can roughly estimate the proportion of fibres cut in a single case. Now, in an ordinary case of severe recent hemiplegia, the plantar reflex on the "sound" side is usually flexor in all positions of the limb, even when the knee-jerk is markedly increased and ankle clonus is present.

But cases are met with in which the pyramidal lesion is sufficient to produce the condition I have described as pyramidal equilibrium on the "sound" side. An examination of the spinal cord of one such case (see Case 5) showed an unusually large number of homolateral fibres degenerated (Marchi method), and the relative proportion was about 1:10; the opposite pyramidal tract was almost completely degenerated as a result of the cerebral lesion. Further, in those cases which are on the flexor side of the equilibrium point in these hemiplegic cases, the proportion of fibres degenerated is about one-twentieth of the entire pyramidal tract in the dorsal region. Again, cases of pyramidal equilibrium on one side only, with the other side showing no appreciable lesion, occur clinically in which one is able to test the relative power of the two sides, *e.g.* in a case of tumour encroaching upon the Rolandic cortex. In these cases, the power on the former side is usually only slightly impaired as compared with the sound side.

Each of the above facts alone is of little value, but conjointly they strongly point to the conclusion that in acute conditions the point of pyramidal equilibrium is reached when about 5 to 10 per cent. of the fibres are thrown out of action; that is to say, if more than 10 per cent. are destroyed an extensor response will

result; if less than 5 per cent. are destroyed, the plantar reflex will still remain flexor.

The point of pyramidal equilibrium is fixed in quite another position in chronic cases, and that more particularly in cases which have been chronic from the outset. In one such case, a patient suffering from primary degeneration of the pyramidal system (Case 13), who, in a state of bi-lateral pyramidal equilibrium, died suddenly of heart-failure, there were about as many degenerated as normal fibres in the pyramidal tracts (Weigert method); here the disease had probably been slowly progressive for eight years.

I recently had an opportunity of examining a large number of hemiplegics who were inmates of a workhouse infirmary. In all of these the response on the "sound" side was flexor; whilst of those whose hemiplegia was of over ten years' duration, many (about 35 per cent.) showed a flexor response on the hemiplegic side, even though the leg was rigid and powerless.

Further examples might be cited to the same effect; but the inference is clear, that in pyramidal disease of chronic onset or of long standing, to reach the point of pyramidal equilibrium there must be a far more extensive pyramidal disease than in the acute conditions. Further, it is clear that it is only when the disease is slowly progressive that the condition of pyramidal equilibrium is maintained for any length of time; the equilibrium is always unstable, and in cases when the paralysis is the result of a non-progressive lesion tends constantly to give way to a permanent flexor response.

#### INFLUENCE OF FATIGUE ON THE PLANTAR REFLEX.

The condition of pyramidal equilibrium is of interest from another point of view. In a healthy adult, repeated stimulation of the sole on each occasion gives rise to a flexor response, and similarly a patient so diseased as to give a deliberate extensor response gives the same response on repeated stimulation: in both cases the intensity of the response diminishes with a constant degree of stimulation, but the character of the response remains constant. But if the plantar reflex of a patient in a state of pyramidal equilibrium be tested in the extended position of the limb, the stimulus being applied to the outer edge of the

sole, I have often found that although the first two or three responses may be extensor, succeeding ones are nearly always flexor; this exhaustion of the extensor response lasts for several minutes and appears to be totally independent of voluntary movement. Hence in case of doubt it is always advisable to observe accurately the result of stimulating the sole the *first* time; if an extensor response then occurs, even though immediately afterwards further stimuli cause a flexor response, it may be safely assumed that we have evidence of organic interference with the pyramidal system.

If an extensor response so obtained is followed by a series of flexor responses, it is of course eminently advisable that the patient should be re-tested after several minutes' interval, so as to be certain that no accidental voluntary movement was introduced on the first occasion. When an extensor response is repeatedly obtained in this way, the test is of extreme delicacy, but is absolutely diagnostic of organic disease. I have never seen a case in which such an extensor response was present where there was reason to suspect merely functional disease. In the appendix, I instance a case in which the reflex was of this character and in which post-mortem evidence was obtained of pyramidal disease (Case 13).

#### CONDITIONS UNDER WHICH THE PLANTAR REFLEX IS OF GREATEST VALUE.

##### 1. *In symmetrical pyramidal disease.*

Seeing that in testing the plantar reflex we do not rely upon the extent or range of any movement, but only upon the character or type of such movement, the plantar reflex is not less useful in symmetrical than in unilateral disease. In my experience, the plantar reflex is always of the flexor type in the healthy adult; but I know of no standard by which we may measure what exaggerations of the knee-jerk shall be considered indicative of organic disease. Even in cases of pyramidal disease of considerable standing one occasionally meets with patients in whom the knee-jerk though brisk is not more exaggerated than is sometimes seen in neurasthenia. In other words, the value of the knee-jerk depends upon quantitative results, and the limit of its excursion in health is difficult to estimate; but the plantar

reflex does not fail in symmetrical disease because the criterion is one of *quality*, not of *quantity*.

2. *In acute diseases.*

As I have before mentioned, the plantar reflex changes its character (from the flexor to the extensor type) coincidentally with the onset of pyramidal disease: it seems to be hardly affected at all by "shock"; on the other hand, the tendon jerks are usually absent in sudden cerebral lesions. In apoplexy, for instance, it is usual for both the knee-jerks to be absent, but the plantar reflexes indicate the side on which the lesion has occurred even though the patient be still unconscious. It must be remembered, however, that a hæmorrhage bursting into the ventricles of the brain frequently gives an extensor response on both sides from the bilateral pressure it exerts.

3. *In tabetic and allied states.*

The plantar reflex usually persists in tabes long after the knee-jerk has disappeared, and remains a criterion of organic pyramidal disease. The same is true of the plantar reflex in peripheral neuritis, in Friedreich's disease, and in sub-acute combined degeneration of the spinal cord.

4. *Focal lesions of the spinal cord.*

In cases of pressure upon the third or fourth lumbar segments, the knee-jerk is absent but the plantar reflex remains intact. In all probability the spinal centre for the plantar reflex is situated in the last lumbar and first sacral segments; so long as these segments, themselves healthy, maintain intact their connections with the skin and muscle there represented, the plantar reflex remains as a reliable guide to organic pyramidal disease.

5. *Hysteria, etc.*

Of all the cases commonly met with in neurology, those in which a diagnosis has to be made between early Disseminate Sclerosis on the one hand and Hysteria and allied conditions on the other are the most important and often the most puzzling. It is in this class of cases *par excellence* that the plantar reflex is of the greatest value. The earliest symptoms of disseminate sclerosis depend upon the part of the nervous system first affected; but in a large majority of cases the pyramidal system is early involved, either in the capsule, mid-brain, pons, medulla or cord. These particularly seem to be the cases in which the symptoms and physical signs may resemble those of functional

paralysis, and it is in every case important to be certain, even if "functional" symptoms are present, whether there is or is not any organic basis for the disease. If an extensor response is obtained, it may be safely assumed that the disease is not purely functional. But the converse is not necessarily true, and it cannot be asserted without further evidence that only functional disease is present, although a flexor response greatly favours such a hypothesis; it must be remembered that the lesions of disseminate sclerosis are often of slight degree, so that such a lesion of the pyramidal tract may not be severe enough to give rise to an extensor response (see Case 12).

#### 6. *Choreas, etc.*

In one other class of cases is the plantar reflex of great value. It is often difficult to say whether a patient with choreiform movements is suffering from cerebral degeneration or not. In Sydenham's chorea the plantar reflex is never extensor; but in those cases of cerebral diplegia with choreiform movements, an extensor response is the rule.

### CAUSES OF DISAGREEMENT AS TO THE VALUE OF THE REFLEX.

Statistics have frequently been published from which authors have concluded that a flexor response often occurs in spastic conditions of the lower extremity, and this has been urged as detracting from the value of the reflex. I have already indicated the conditions under which such a flexor response is obtained. The essential contention is that the presence of the extensor response indicates organic disease. Much more important are those results in which an extensor response is said to have been present apart from organic disease.

As I have already pointed out, it is of primary importance that the conditions for the examination of the reflex should be as good as possible. It is easy to test the knee-jerks of a fully-dressed patient as he sits in a chair, but it is much more difficult to determine the nature of his plantar reflexes. I would insist that in every doubtful case *the patient should lie down and that a minimal stimulus should be applied to the sole whilst his attention is distracted and the joints are supple*. Only by these means is it possible to be certain that the result is a reflex not mixed with voluntary movements. Further, the *first* movement occurring at

the metatarso-phalangeal joint of the big toe should alone be watched for; the second movement may be of wider range in an opposite direction, but is often the result of voluntary movement—in any case it should be disregarded.

But different methods of obtaining the reflex cannot account for all of the observations in which an extensor response is said to have occurred apart from organic disease. For instance, two cases have recently been recorded by Harris (*Review of Neurol. and Psych.*, 1903) in which an extensor response occurred—bilateral in one case, unilateral in the other—and where concurrent symptoms and complete cure lead the author to consider the condition to be purely functional. Where, then, is the fallacy? I would suggest that in both cases there was true organic disease as a basis for the conditions. In the first case Harris freely admits the possibility, but rejects such a diagnosis on the ground of complete recovery. I would like to compare this case with the one I record (Case 9).

In my case, besides the double extensor response there were present two other definite signs universally regarded as the result of organic disease, viz., diplopia and incontinence of urine; but is it reasonable to say, because this patient recovered completely, that therefore his condition was merely functional? Compare this again with case in which a rigid paraplegia with typical extensor responses was cured by removal of a tumour pressing upon the spinal cord (Case 3). Here, again, a slow recovery occurred, and when seen about eight months later, no sign of organic disease could be found; here, of course, no one could deny the presence of an organic lesion. I think we are apt to underrate the power of repair of the central nervous system, especially in people under middle age. The fact of complete recovery occurring does not, in my opinion, justify a diagnosis that no organic disease has been present.

The second case recorded by Harris is again not convincing. As before, only clinical evidence is available, and the full description of the patient at the acme of his illness—when the double extensor response was present—strongly suggests an inflammatory condition of the brain or meninges, in both of which conditions extensor responses might occur without shaking one's faith in the value of the plantar reflex. I am quite aware that the case is not one typical of meningitis, but equally it is not typical

of any functional condition. Again, I would ask for comparison of his case with another which I record (Case 10). In my case, the presence of Jacksonian convulsions, of optic neuritis, and of incontinence of urine may, I think, be taken as definite signs of organic disease, but otherwise the two cases bear a close resemblance to one another, particularly in that they both recovered completely after exhibiting a double extensor response. I should therefore be inclined rather to consider that both of the cases recorded by Harris had an organic basis, and I do not think that he has proved that the extensor response has occurred apart from organic disease.

I have dealt at some length with these cases because they and the method of examination are fully recorded by an investigator whose accuracy of observation cannot be questioned. I believe that such cases constitute a large majority of those which are supposed to be exceptions to the rule, and I think that it will ultimately be found, not that they tend to nullify the value of the extensor response, but that the extensor response in these cases will give a still further insight into the nature of some obscure nervous conditions.

It will be noticed that in recording above the results of my observations, I have not given any statistical record. I have done this deliberately because I do not believe they serve any useful purpose. The cases vary so much—in duration and in severity—that the point of what I have called pyramidal equilibrium must vary from case to case, so that, for instance, one case of hemiplegia will give an extensor response, and another, of different duration and severity, will give a flexor response. I have endeavoured rather to analyse the reasons for the response in each case, and to record the summary of my observations illustrated by particular cases which seem to throw especial light upon the point at issue.

#### CONCLUSIONS.

1. In adults, an extensor response (Babinski's sign) never occurs in health; it is always indicative of organic disease.

The pyramidal system need not be so injured as to show post-mortem lesions. An extensor response may be produced in any condition which greatly raises intracranial pressure, even if

that condition does not cause a demonstrable lesion of the pyramidal system. For instance, an extensor response may occur in hydrocephalus, meningitis, cerebral tumour, etc.; in such cases the extensor response usually only occurs in comatose or semi-comatose states.

An extensor response also occurs in convulsive conditions of organic origin, *e.g.* in epilepsy, uræmia, infantile convulsions, strychnine poisoning, etc.

An extensor response never occurs in hysteria and allied conditions unless some organic disease is also present.

2. A flexor response is the normal plantar reflex of the adult; it may occur also in pyramidal disease: (a) in acute conditions if only a small amount of the pyramidal influx is removed by an organic cause; or (b) in chronic cases, even when a large amount of the pyramidal influx is removed.

3. Under certain conditions, it is sometimes possible to obtain in the extended position of the patient an extensor response, and in another position (Collier's) a flexor response from the same foot; this may be termed the position of pyramidal equilibrium.

4. The position of pyramidal equilibrium is lower in the scale of pyramidal disease in recent and acute cases than in cases where the disease is of long standing or of very chronic origin.

5. There is a constant tendency in the adult for an extensor response to give way to a flexor response; the more chronic the case and the less severe the pyramidal lesion, the more rapidly will a flexor response be re-established, even though the limb remain severely paralysed and contracted.

6. The infantile response of children under the age of two years is dependent upon the immaturity of the pyramidal tracts; it resembles the response of pyramidal equilibrium rather than that of a full spasticity.

7. The extensor response comes on *simultaneously* with the incidence of pyramidal disease. The reflex is not abolished by total transverse section of the cord, and appears to be less affected than any other reflex by "nerve shock."

8. The plantar reflex may be absent in health when the foot is cold or damp; it is also frequently absent in hysteria, multiple neuritis, infantile paralysis affecting the leg, and in severe *tabes*.



## APPENDIX.

The cases recorded here are brought forward to demonstrate abnormal or unexpected ways in which the extensor response may be developed, or to illustrate special points in the foregoing article. Most of them are unusual cases—cases in which, without full and careful examination, it might have been supposed that the extensor response was present in the absence of organic disease of the nervous system. In Cases 9 to 12 clinical examination alone was possible, but concurrent signs and symptoms sufficiently indicate that organic disease was present. In the other cases, post-mortem or operation evidence was relied on as determining the nature of the disease.

For the purpose of comparison, I reprint the description of Harris's cases side by side with similar ones of my own.

CASE .I. Occurrence of extensor response immediately after a lesion of the pyramidal cortex.

George C., a bank cashier, admitted to hospital on October 10, 1901, complaining of headache, vomiting and diplopia of three months' duration.

*Family history* and *past history* good.

Present illness began on July 15th, 1901, with a "bilious attack." This recurred a fortnight later, and again six weeks afterwards, and with this attack diplopia came on and has persisted ever since. Frequent headaches and vomiting for the last three weeks. Has had one attack of transient aphasia lasting ten minutes, but no convulsion.

On admission, he was a tall, thin, well-educated man. Headache was general and persistent, and he vomited occasionally. There was intense double optic neuritis, but vision was normal in each eye. There was slight paresis of the left sixth nerve.

No other paralyses or localising symptoms could be detected. A trephine was decided upon to save the eyesight, and on Nov. 15th, 1901, he was trephined over the right frontal region, a piece of the parietal bone the size of a five-shilling piece being removed and the dura being opened. The brain immediately bulged forwards into the wound, and it was seen that the Rolandic cortex was partly involved.

Immediately after the operation, as he was recovering from the anæsthetic, the plantar reflexes were tested; it was now found that the left plantar reflex was of the extensor type, although immediately before the operation it had been flexor; the right plantar remained flexor. After the operation the cerebral hernia rapidly increased in size, and the reflexes remained one flexor and the other extensor, until his death from asthenia on April 18th, 1902. The optic neuritis had ceased to progress—had indeed rapidly subsided—after the operation.

At the post-mortem it was found that the tumour was in the left frontal region; the hernia contained brain tissue only. Probably the hemiplegia and extensor response were caused by the Rolandic convolutions being squeezed through the opening in the skull and becoming strangled at this point.

**CASE II.** Complete section of the cord. One knee-jerk just present, both plantar reflexes well-marked and of the extensor type.

Frederick G., aged 25, trooper, was admitted to the National Hospital, Queen Square, in May 1902.

*History.*—Whilst in a half prone position, in the act of firing, he was shot by a Mauser bullet, which entered near the left shoulder and passed out at the opposite loin. His legs were instantly completely paralysed, and have remained so ever since.

Examined six months later, he was found to have complete motor and sensory paralysis below the fifth dorsal segment. The left knee-jerk was absent, but the right was feebly though certainly present. The plantar reflexes were both well marked and of the slow extensor type.

At the patient's urgent request, laminectomy was performed with the intention of suturing the severed cord if possible. It was found, however, that the cord had been completely cut through, and that the ends had retracted until they were over two centimetres apart. The wound healed by first intention; no further change occurred in his condition.

**CASE III.** Compression of the spinal cord by a non-malignant tumour, causing complete paralysis of both lower ex-

tremities, with extensor plantar reflexes; removal of tumour, followed by complete recovery of power and return of flexor responses.

Jacob F., aged 25, shoemaker, was admitted to the National Hospital, Queen Square, in May 1902.

Family history and past history unimportant; had never had syphilis.

The onset of the present illness was insidious, beginning with weakness of the right ankle seven months previously. The right knee next became weak, and the left leg was affected shortly afterwards. Some hesitation in micturition, no other sphincter difficulty. For three months has had girdle sensation of tightness around the lower abdomen. No pain or other sensory symptoms.

On admission, was well-built, with no constitutional disturbance. There was no affection of any cranial nerve, and the arms and the upper part of the trunk were normal in all respects. Both lower extremities were almost completely paralysed, but not wasted; they were very rigid, with knee clonus and ankle clonus. The plantar reflexes were of the slow extensor type. The lower muscles of the abdomen were also partly paralysed (below the eighth dorsal supply). There were no sensory changes.

By July 1902 the paralysis had become complete in the lower extremities, and there were sensory changes throughout below the eighth dorsal supply. There was no sign of vertebral disease.

On Aug. 26th, laminectomy was performed; an oval myxofibroma, the size of a walnut, was found pressing on the dura and enclosed spinal cord. It was removed with some difficulty, and the cord was felt to be much flattened at this point.

Recovery was uninterrupted; he regained some movement in his lower limbs within three days, by which time all sensory disturbance had disappeared. His legs gradually regained power, and three months later was able to walk without assistance, but the legs were still stiff, with extensor reflexes and much increased knee-jerks. By Feb. 1903 he was walking perfectly; the knee-jerks were still increased, but the plantar reflexes were now of the flexor type.

CASE IV. Hydrocephalus in an adult, with double extensor response.

Gertrude L., aged 19, shop-assistant, was admitted to the National Hospital, Queen Square, on May 29th, 1901.

*Family history.*—Unimportant.

*Past history.*—Has never menstruated. No severe illness.

*Present illness.*—Has suffered from headache, vomiting and loss of sight during the last eighteen months, especially lately. Gradually increasing drowsiness for three weeks.

*On admission.*—She lies in bed on her back in a semi-comatose state, answers correctly simple questions when roused, but volunteers nothing. Intense double optic neuritis, passing into atrophy. Marked nystagmus. No squint. No paralysis nor anaesthesia could be made out.

*Reflexes.*—K. J. equal, increased. Ankle clonus on both sides. Plantar reflexes both of the extensor type.

She died comatose two days later.

*Post-mortem.*—There was no cerebral tumour present; there was an intense hydrocephalus, the lateral and fourth ventricles being enormously distended. No cause for the condition could be discovered.

CASE V. Severe hemiplegia (embolic) with *double* extensor response; unusually large number of homolateral pyramidal fibres degenerated.

Emily E., machinist, aged 41, admitted to the Queen's Hospital, Birmingham, on Sept. 18, 1903, complaining of shortness of breath of four years' duration. Had had an attack of acute rheumatism at the age of 27. On admission was found to have signs of mitral and aortic disease. There was no sign of organic nervous disease, and both plantar reflexes were of the flexor type.

On October 14th she had a sudden attack of severe left hemiplegia; there was much mental disturbance, but not total unconsciousness. It was impossible to test the power of the right side. Reflexes: all arm-jerks active (about equally); right knee-jerk much increased; left active, but less than right; left plantar reflex of slow extensor type, whilst stimulation of the right sole causes sharp extension of the hallux *in all positions*

*of the limb.* After a few days pyramidal equilibrium became established on the right side, *i.e.* a flexor response could be obtained when the limb was in the flexed position, and a sharp extensor response when the limb was fully extended. This condition persisted until her death on Nov. 25th.

*Post-mortem.*—Mitral, aortic and tricuspid disease were the heart lesions present. The right middle cerebral artery was blocked at its commencement by an embolus, and the corresponding part of the cerebrum was softened and shrunk. There was no lesion on the left side, nor could any other focus be found in the crura, pons, medulla or cord. Stained by the Marchi method, the degeneration stood out with great clearness; the whole right pyramid was degenerated, and at the decussation gave off an unusually large number of (uncrossed) fibres to the lateral column. The anterior pyramid was of normal size.

CASE VI. Uræmia with double extensor response. Scattered pyramidal degeneration found post-mortem.

Rose B. was admitted to the Queen's Hospital, Birmingham, on Dec. 30th, 1903, suffering from dropsy of four days' duration. Family history unimportant. About six weeks before admission she developed a red rash on the chest, but no other symptoms suggestive of scarlet fever were noticed. The rash died away in a few days, but three weeks later it was noticed that the urine was thick, and that it became deficient in quantity. Headache and puffiness about the face and legs came on, and the day before admission she had a convulsion. A second fit occurring the next day, she was brought up to hospital and was admitted.

On admission, she was found to be a well-built girl, sixteen years of age. There was general anasarca. She passed an average of ten ounces of urine a day, the urine being loaded with albumen, and containing many casts and much renal debris. There was marked and continuous headache, but no vomiting.

Shortly after admission she had a series of general convulsions, followed by a period of semi-coma. During this quiescent stage the plantar reflexes were found to be of the full extensor type, the knee-jerks being absent. Despite treatment, the convulsions recurred, and she died on Jan. 10th, 1904.

*Post-mortem.*—There was a general anasarca present; the

kidneys were large, and showed an intense interstitial and tubular nephritis. There were no naked-eye changes in the spinal cord, but the cerebral cortex was enormously congested. On staining the cord by the Marchi method, slight scattered degeneration was found in the lateral and posterior columns.

**CASE VII.** Tubercular meningitis, simulating typhoid fever ; double extensor response.

A boy aged 12, under the care of Dr Foxwell, sent in to the Queen's Hospital, Birmingham, from an industrial school. The only history obtainable was a vague one from the boy himself, to the effect that he had been ill for about ten days with frontal headache and malaise. He was found to have a temperature of 102°, pulse 80, respiration 18 ; the face was pale, the tongue furred and rather dry. There was no enlargement of the spleen, and no rose spots ; he was slightly constipated. There was no optic neuritis ; there was no rigidity of the limbs ; the knee-jerks and the ankle-jerks were brisk, but no ankle-clonus was present. Stimulation of the outer surface of the soles gave a quick extensor response on both sides, whereas stimulation of the inner surface of the soles gave marked flexion. During the following week I repeatedly tested the plantar reflexes, but always with the same result. The lad then died in convulsions, and at post-mortem was found to have tubercular meningitis and general acute tuberculosis. When this boy was first seen, the clinical condition closely resembled that of enteric fever, but the extensor response could not be explained on this basis. The post-mortem justified the conclusion of organic intra-cranial disease.

**CASE VIII.** Tertiary syphilis with cirrhosis of kidneys ; repeated hæmorrhages. No nervous symptoms, but double extensor response and absent knee-jerks. Degeneration of the spinal cord proved post-mortem.

Ralph W. L., a porter, aged 36, was admitted to the Queen's Hospital, Birmingham, on Aug. 6th, 1903, complaining of spitting blood and passing blood in his urine.

*Past history.*—Had had syphilis (untreated) ten years before. Frequent severe headaches recently. For four months past

has had recurring attacks of hæmoptysis, hæmatemesis hæmaturia and melæna. Has grown steadily paler and weaker.

*On admission.*—Muscular, but pale and tired-looking. Arteries generally thickened, blood-pressure high. No signs of organic heart or lung disease. Urine contains a heavy cloud of albumin and some tube-casts. Well-marked albuminuric retinitis. No nervous signs or symptoms.

*Progress.*—The hæmorrhages continued, despite treatment, and he died of anæmia on Sept. 4th, 1903. He was carefully re-examined on Aug. 30th, and it was then found that although there was no *symptom* pointing to disease of the nervous system, yet the knee-jerks were absent, and the plantar reflexes were of the quick extensor type. He had no numbness, tingling or anæsthesia, nor could any paralysis be made out, although the general weakness was extreme. These nervous signs persisted in the absence of any symptom until his death.

*Post-mortem.*—Contracted granular kidney and secondary vascular changes. No macroscopic anomaly in the nervous system. Sections of the spinal cord stained by the Marchi method show much recent degeneration scattered throughout all columns. In the pyramidal tracts the degenerate fibres constituted about one-eighth of the entire number.

CASE IX. Acute attack of (?) disseminated myelitis, with double extensor response. Complete recovery.

A. K., a man aged 24, was admitted to the National Hospital, Queen Square, on April 17th, 1902.

*Complaint.*—Numbness of legs and abdomen, fourteen days; diplopia, incontinence of urine and difficulty in walking, three days.

*Family history.*—Unimportant.

*Past history.*—Had rheumatic fever at age 17, and has suffered from occasional rheumatic pains ever since. No venereal disease.

*Present illness.*—Onset with pain in the right calf four weeks ago. A fortnight later the pain in the calf gradually merged into a numbness which soon spread upwards to the hip and then involved the other leg. For two days has been unable to feel his motions when defæcating, but has had no accident.

Diplopia for distant objects has been constant for three days. Hesitating micturition and occasional incontinence of urine for three days.

*On admission.*—Muscular man, viscera healthy. Face flushed, sweats profusely; temperature 99·6°, pulse 92.

*Nervous system.*—Both optic discs cedematous and congested. Pupils large, equal, react well. No nystagmus; no obvious ocular palsy, but diplopia on looking at distant objects. No weakness or tremor of arms. Legs generally weak but not stiff. Numbness up to umbilicus on trunk, of both legs, and of four fingers of the right hand. Over these areas sensibility to heat, cold, touch and pain is slightly diminished. Can just walk alone; gait feeble and ataxic.

*Reflexes.*—Arm-jerks all very brisk. Epigastric, normal. Abdominal, equal, but diminished. Knee-jerks, equal, much increased, clonus both sides. Ankle clonus present on both sides. Plantars, right, slow extensor response; left, quick extensor.

*Progress.*—By June 11th all symptoms had disappeared and he was walking well; ankle clonus and extensor responses still present. On Sept. 10th he was quite well, and the plantar reflexes were now both flexor in type; ankle clonus absent.

CASE IX. A. Dr Harris's Case 1, as published in *Review of Neur. and Psych.*, Vol. i., page 326. This case is reproduced for comparison with my own case, as explained in the text.

J. S., a hawker, aged 25, a small, ill-developed man, was admitted to St Mary's Hospital, October 29th, 1901, under Dr Cheadle. He was admitted for paralysis of both legs and loss of voice. Twelve months previously, when walking in the street, his legs suddenly gave way under him, and he fell down. For eight weeks he had pins and needles sensation in both legs, when the pains suddenly left him and the legs became numb. Since then, sensation has completely returned, but he has been quite unable to use his legs, and he gets about on crutches. Never any bladder trouble or girdle pain. A week before admission he completely lost his voice. On examination in bed the legs seemed strong for all movements, but his attempts to



walk were suggestive of "functional" gait, the legs being held stiffly, and dragged about with apparent great effort, not at all suggestive of a true spastic gait. There was no anæsthesia. Knee-jerks brisk and equal; no ankle clonus. There was bilateral brisk extensor plantar reflex, both great toes being briskly extended, without any dorsiflexion of the ankle, but slight flexion of the small toes. He was completely aphonic, due to functional adductor paralysis, the cords lying wide apart, with very slight movement only on attempts at phonation, though he could adduct them perfectly in the act of coughing. The eye movements were normal, and the visual fields were not contracted. The diagnosis of functional paralysis and aphonia was made, with the reservation that there was probably some organic spinal disease behind it, on account of the typical extensor plantar reflex. He was sent down to me in the electrical department, and I applied strong faradism with a wire brush for about five minutes, suggesting to him at the same time that it would cure him. The effect was immediate, and he was able to walk moderately well upstairs to the ward, carrying his crutches over his shoulder. The aphonia was also cured and did not return. His gait improved day by day until, at the end of a week, he could walk perfectly. The extensor plantar reflex persisted unaltered for a few days, but at the end of a week it was found to be of the normal flexor type, and remained so. He was seen again in July 1902, but there was no recurrence of the paralysis or aphonia, and the plantar reflexes were still flexor.

CASE X. Presumably cortical meningitis (? influenzal) in an adult. Convulsions, incontinence of urine, double extensor response, optic neuritis; complete recovery.

Miss R. M., a single lady, aged 32, was seen in consultation first on December 31st, 1903. The family history was unimportant.

*Past history.*—Had been subject to attacks of migraine since childhood; they were prone to occur every two or three months, and usually incapacitated her for about twenty-four hours; they were always quickly relieved by a combination of bismuth and calomel. Had never suffered from fits.

The *present illness* began on December 26th with what appeared to be one of usual attacks of migraine—nausea, vomiting, general headache and lassitude being the most marked features. Calomel gave her relief, and next day she was able to go about as usual. The following day, however, she did not get up at the usual hour, and when looked for was found lying on the bed in a semi-nude condition and unable to explain what had happened. She was kept in bed, and late in the afternoon she had a severe epileptic fit. The fit began as a Jacksonian seizure in the right side of the mouth, spread to the arm and leg, and then became general. The fit was followed by stertorous breathing for some minutes, and then she went to sleep. There was no biting of the tongue nor incontinence of urine. On waking up two hours later she was unable to speak, but seemed to understand what was said to her. During the next two days she gradually became worse; two more major attacks and several minor ones occurred. She seemed to suffer from headache, but was unable to speak, and the left arm became powerless. When I first saw her on December 31st, she lay on her back in bed unable to speak; her only reply to all questions was to put out her tongue, but she followed one's movements about the room in a way which suggested some intelligence. Occasionally she would put up her right hand to her head as though she were suffering from headache. The left arm was not moved at all, even when the right was held down. There was no optic neuritis nor vomiting, and food was taken well. There was no squint, and the eye movements seemed normal. No signs of paralysis could be detected in the face or tongue. Both legs were occasionally moved. The knee-jerks were normally brisk, and the plantar reflexes were of the quick extensor type. Urine was passed involuntarily.

On January 3rd, 1904, she was again seen. She had wasted rapidly and was now fully comatose, with Cheyne-Stokes breathing. There had been no more fits, possibly on account of the bromide which she had taken since the previous visit. The pupils were small, equal, and still reacted to light feebly; there was early optic neuritis in the left eye (the right was not seen). No voluntary or reflex movements of the limbs were made, and food was taken with difficulty. The knee-jerks were now very brisk, and the plantar reflexes were both of the full extensor type.

From this point onwards she steadily improved. No further fit occurred, consciousness and power in the limbs gradually returned, and a month later she had completely recovered except for some general weakness. The plantar reflexes were now of the flexor type, and the knee-jerks were normal. There was no sign of pneumonia or tubercle throughout. Recovery was complete by March 1904.

The case was thought to be one of cortical meningitis, probably due to the influenza bacillus.

CASE X. A. Dr Harris's Case 2. This is reproduced for comparison with my case, as explained in the text.

W. W., a boy of 13, was admitted to St Mary's Hospital on June 30th, 1903. On June 23rd he was carrying coals upstairs with his left hand when he felt a sudden numbness and loss of power in the left hand and arm, and numbness down the left leg and side. He dropped his burden, supporting himself by the balusters. He walked home, and then involuntary twitching of the left arm, from the shoulder downwards, commenced, occurring three or four times every hour. Next day the arm was weaker and the twitching worse, the leg remaining the same. On the 25th the arm was worse, and the leg became weak, so that he limped, the numbness on the left side also being worse. The following day he could not walk without support, and he was still weaker. From the 26th till the day of admission he remained the same. No headache or sickness, and he stated positively that previous to the attack he had felt as well as ever since he had had scarlet fever fifteen months before, followed for a short time by bilateral otorrhoea. On examination there was weakness of all movements of the left arm and leg, with hemianæsthesia to the mid-line, involving the left arm and leg and left side of the body below the level of the clavicle and just above the shoulder. There was no anæsthesia of the head or neck, and it was less complete below the knee. Involuntary spasmodic twitchings of the left arm were frequent, especially when under observation. The knee-jerks were equal and normal, but there was fairly well marked left ankle clonus and typical bilateral extensor plantar reflex, brisk extensor movement of the great toes, without any dorsiflexion of the ankle. The optic discs,

eye movements and visual fields were normal. No pyrexia. Two days after admission he complained of frontal headache and was sick once. He looked distressed, and lay curled up in bed. On the 5th of July the headache had gone, and the anæsthesia disappeared from the left leg and arm, but still present on the left side of the trunk. Still well marked bilateral extensor plantar reflex. On the 7th there was complete flaccid paralysis of the left arm and leg, and the anæsthesia had returned, with complete loss of sense of position of the arm and leg. There was no diaphragmatic breathing, respiration being entirely costal and by the accessory muscles. On the following day the anæsthesia had again disappeared, but the breathing remained as on the 7th. Left ankle clonus and extensor plantar as before. No optic neuritis. On waking on the morning of the 12th he found he had power to move his left arm and leg. The power gradually increased, and in a few days he was walking perfectly, all the signs of weakness and anæsthesia having disappeared, and he was discharged cured July 22nd. The extensor plantar had now changed to the flexor type, and when seen recently, April 28, 1903, he had remained perfectly well, and there was no sign of weakness or anæsthesia, the plantar reflexes being now brisk flexor in type.

CASE XI. An attack of acute anterior polio-myelitis in an adult, resulting in paralysis of one leg : extensor response on the "sound" side.

Henry S., seaman, aged 21, was admitted to the National Hospital, Queen Square, on December 12, 1901.

*History.*—Whilst running on deck on August 8th, 1901, he had a sudden pain in the right calf which caused him to fall down. He walked with difficulty to his bunk, and stayed there. Next day the right lower extremity was powerless and the left leg and both arms were weak. After a few days only the weakness of the right leg remained.

On admission four months after the onset, he was found to have an atrophic paralysis of the right lower extremity; the only muscle below the hip in which the slightest trace of power remained was the tensor fasciæ femoris. The lower erectores spini and the left thigh muscles were thought to be atrophic and weak,

but there were no electrical changes here. There was no spasticity of the left leg, and no sensory changes were anywhere present.

*Reflexes.*—All absent in right leg.

Left K. J. very feeble.

Left ankle-jerk very brisk, but no clonus.

Left plantar reflex well marked extensor in type.

The condition underwent no appreciable change during his stay in hospital. It was supposed that he had had an attack of acute anterior polio-myelitis, which about the lumbar region of the cord had to a slight extent involved the lateral column on the left side, thus giving rise to an enfeebled knee-jerk corresponding to the seat of the lesion, and to slight signs of spasticity—an increased ankle-jerk and a plantar reflex of the extensor type—in the muscles represented below the lesion.

**CASE XII.** Flexor plantar reflexes occurring in an early stage of disseminate sclerosis.

William L., aged 28, admitted to hospital in July 1899 with difficulty in walking.

Family history unimportant. Had had two severe falls—one in 1885, and the other in 1895. The present illness is dated from this fall. Staggering gait and precipitate micturition have been his chief complaints since then.

On admission, he was found to have weakness of all four limbs, especially marked on the right side. The deep reflexes were all exaggerated, but the plantar reflexes were of the flexor type.

When seen again two years later, he was much worse. He had developed nystagms, optic atrophy, and further weakness of the limbs. Incontinence of fæces occurred at times. The deep reflexes remained exaggerated, but the plantar reflexes were now of the well-marked extensor type.

**CASE XIII.** Primary pyramidal degeneration, with “pyramidal equilibrium,” simulating hysteria. Diagnosis confirmed post-mortem.

A single woman, aged 53, was admitted to the National

Hospital for the Paralysed and Epileptic on July 25th, 1902, under the care of Dr Hughlings Jackson.

*Complaint.*—"Hysteria"; laughing without reason, loss of power of walking, loss of power of speaking properly.

*Family history.*—Quite good.

*Previous health.*—Always good. Menstruation began at 13; climacteric at 43. No exceptional strain, physical or mental. Ten years ago had a fall on to her back, in which she had pain for a few days afterwards. She dates her illness from this fall.

*Present illness.*—Onset insidious. She began to find difficulty in controlling herself and in replying sensibly to questions: began to laugh at trifles, independently of thinking the events laughable. For two years has found walking becoming more and more difficult. Latterly, also, has found difficulty in pronouncing words.

*State on admission.*—Short, rather stout woman, dark grey hair. Memory fair, answers questions well, but often bursts out laughing, and when asked why she does so, can give no reason. No crying attacks. Sleeps well. The face has rather a stiff look, suggestive of Parkinson's mask. Power in arms and legs symmetrical, probably below average. Attitude and gait resemble to slight extent those of paralysis agitans; there is no tremor.

*Reflexes.*—Arm and knee jerks all brisk, no ankle clonus. The soles of feet are very "ticklish." With knee and hip bent (Collier's position), plantar reflexes invariably flexor. In extended position with stimulus applied to outer edge of sole, the first response is quick extensor movement of the hallux; subsequent stimulations within the next few seconds always resulted in a flexor response. This result was repeatedly obtained on later examinations.

No appreciable change occurred in the nervous condition or in the reflexes. She died quite suddenly on November 23, 1902, of heart failure.

*Post-mortem.*—There was extensive bilateral pyramidal tract degeneration (Weigert method): there was no sign of recent disease of the spinal cord.

---

## A PRELIMINARY NOTE ON AN ABERRANT CIRCUM-OLIVARY BUNDLE SPRINGING FROM THE LEFT PYRAMIDAL TRACT.

By G. ELLIOT SMITH, Cairo.

(With 4 Figures.)

DURING the last four months I have seen no less than fifteen brains in which a large bundle of pyramidal fibres dissociated itself from the rest of the (left) pyramid, and wended its way around the olivary body to the region of the restiform body (Fig. 1). I shall call it the "*fasciculus circumolivaris pyramidis*."

The frequency of its occurrence here cannot be explained on racial grounds, because my fifteen cases included the brains of Egyptians (seven cases), Soudanese negroes (four cases), Turks (two cases), a Greek, and a man from Bokhara. Considering this wide racial distribution, it seems highly probable that this aberrant bundle must often occur in the brains of other Peoples. It has probably escaped the attention of most anatomists because it does not often happen that the medulla oblongata is submitted to careful examination in large series of fresh brains from which the pia mater has been removed; and in sections this bundle might readily be mistaken for a group of external arcuate fibres.

In his interesting investigation of the degenerations which were present in five cases of hemiplegia, Stanley Barnes has described one example of this peculiar bundle in these words:—"Another curious set of homolateral fibres was given off from the pyramid just above the middle of the olive (Case 5, Fig. 2). They passed upwards and outwards as a thick band of degenerated fibres round the olive with the external arcuate fibres to reach the region of the restiform body, around which they become scattered, the most prominent bands of fibres being situated near the nucleus cuneatus; two small bundles, however, were almost superficial, and lay close to the floor of the fourth ventricle, and could be traced nearly to the hypoglossal nucleus" (*Brain*, vol. xxiv., 1901, pp. 490, 491). Further on (p. 499) he adds, in reference to this aberrant bundle: "I have been unable to find any reference to a similar tract in the literature."

However, I find the following references to such a bundle in Edinger's and Wallenberg's "Bericht über die Leistungen auf dem Gebiete der Anatomie des Centralnervensystems während der Jahre 1899 und 1900":—"Nach Rothmann (*Arch. f. Psych.*, xxxiii., p. 292, 1900) bestehen Verbindung der Pyramiden mit den gleichseitigen *Fibrae arcuatae externae* und mit der gekreuzten Interolivarschicht. Probst (*Mon.-Schr. f. Psych. u. Neurol.*, vi., p. 91, 1899) sah aberrirende Bündel zur vorderen Grenzzone ventral von der Commissura ventralis des Halsmarkes, ein gekreuztes und ein gleichseitiges 'accessorisches Pyramidenbündel,' das von der Oblongatapiramide via *Fibrae arcuatae externae* ventrales theils zum Seitenstrangkern gelangt, theils caudalwärts umbiegend zwischen Gowers'schem Bündel und Kleinhirnseitenstrangbahn abwärts läuft und Fasern zum Strickkörper abgibt: ferner 'accessorische Schleifenfasern' zu den motorischen Hirnnervenkernen, endlich ein rückläufiges Pyramidenbündel von der Kreuzungstelle zur Region des Quintuseintritts derselben Seite, der Lage nach dem Pick'schen Bündel entsprechend" (p. 42).

Without an opportunity of studying Mingazzini's memoir, "Ulteriori ricerche intorno alle fibre arciformes ed al raphe della Oblongata nell' uomo" (*Internat. Mon.-Schr. f. Anat. u. Physiol.*, xx., 4, p. 105), I am unable to decide whether the statement contained in Edinger's "Bericht" for 1893 and 1894 that the third series of constituents of the restiform body are "*Fibrae afferentes*, von *Fibrae restiformales* gebildete die sich zum grössten Theile in der Raphe kreuzen und sich in die Pyramiden begeben (pyramidale Portion)" (p. 35) refers to the circumolivary bundle.

The presence of circumolivary fibres, which extend across or below the olivary body as far as the lateral columns, has been recorded by many writers [see Edinger's reviews (*op. cit. supra*) for the last ten years]. An excellent example of such a bundle demonstrated by the method of Marchi is to be found in Figs. 383 and 384 of Déjerine's "Anatomie des Centres Nerveux," Tome 2<sup>ième</sup>, 1901, p. 547 (taken apparently from a memoir by Long, "Les voies centrales de la sensibilité générale," *Thèse de Paris*, 1899, p. 224); another case is shown in Fig. 387 of the same work. M. and Mde. Déjerine make the following statements in reference to these "superficial homolateral pyramidal fibres":—"Daus la région bulbaire moyenne et inférieure,



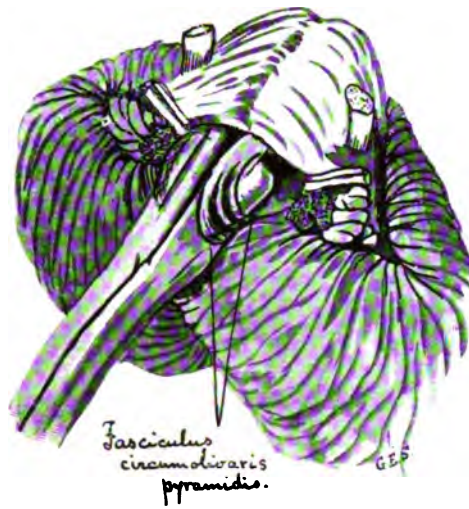


FIG. 1.

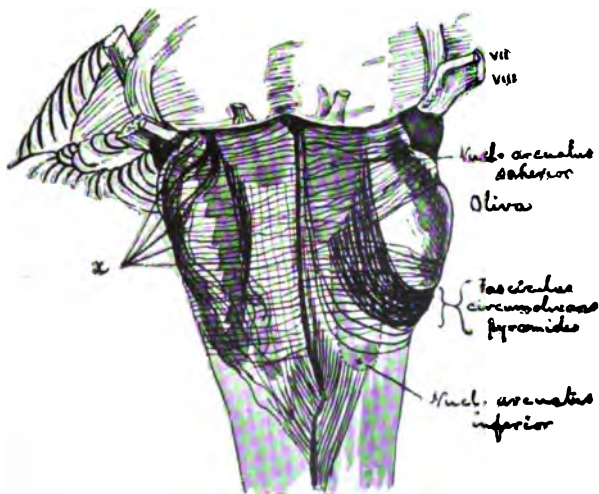


FIG. 2.



la voie pédonculaire abandonne parfois au cordon latéral homolatéral de la moelle un petit nombre de fibres aberrantes qui se groupent en fascicules, affectant un trajet superficiel: les unes (Figs. 383, 384) contournent l'olive bulbaire à la manière des fibres arciformes superficielles, les autres (Fig. 387) s'infléchissent au-dessous de l'olive, toutes deux descendent dans le cordon latéral de la moelle, soit en arrière de l'olive bulbaire (Figs. 383, 384), soit en avant de la corne postérieure (Figs. 387 à 389). Leur présence au devant de l'olive a été signalée par Russell, Spiller, Long, Pick, v. Gehuchten. Elle représentent à notre avis de véritables *fibres pyramidales homolatérales superficielles*, tout à fait comparables aux *fibres pyramidales homolatérales profondes*" (pp. 549, 550).

My series of cases exhibits a bundle of fibres which differs from Déjerine's "superficial homolateral fibres" in the fact that it extends beyond the lateral columns into the restiform body. Nor can it be described as "un petit nombre de fibres," seeing that it is a large prominent cord sometimes as much as one-fifth the size of the pyramid. One noteworthy distinctive feature of the circumolivary bundle in all of my cases is the fact that after reaching the outer border of the olive it proceeds obliquely *upward* and backward. In some cases the inclination upward is so pronounced that the bundle forms a prominent rope-like, U-shaped loop, the two extremities of which are situated alongside the superficial origin of the abducens nerve and the tuberculum acusticum respectively: in other words, the tract is distinctly recurrent.

The fasciculus circumolivaris dissociates itself from the rest of the pyramid immediately below the pons and pursues its downward (caudal) course in front (on the ventral aspect) of main mass of the pyramid. It then curves outward and backward (dorsally) as a large prominent cord slung round the lower end of the olive (Fig. 2) or as several strands crossing the olive (Fig. 1). It then passes obliquely upward and backward, the degree of obliquity of this part of its course being subject to a considerable amount of variation: but in all cases it reaches as far as the restiform body, where its fibres behave in different ways in the various cases that I have examined. In most cases a small bundle of fibres can be seen proceeding inward on the surface of the restiform body to enter the floor of the fourth

ventricle. (In the case represented in Figs. 3 and 4 this bundle, *b*, was exceptionally small.) In every case some fibres (in most instances the vast majority) pass upward in the same direction as the inferior peduncle of the cerebellum, and disappear from view in the lateral recess of the fourth ventricle by passing under the tuberculum acusticum or, in some cases, into the trigonum acusticum. It is impossible to speak with any degree of certainty as to the ultimate destination of these fibres. In some cases they have every appearance of passing underneath the acoustic nuclei to enter the cerebellum: in other cases some of them seem to proceed toward the nucleus of the facial nerve.

It is difficult to believe that any pyramidal fibres pass direct to the cerebellum, such as seems to be the case in several of these specimens. In this connection it is interesting to recall that in one of Barnes' cases there was a small tract of degeneration in the cerebellum with no obvious lesion to account for it: but Barnes states that these fibres "certainly have no connection with the pyramidal degenerations" (*op. cit.* p. 472).

In some of my cases a certain proportion of the fibres of the circumolivary bundle dipped into the substance of the bulb at the lateral edge of the restiform body and could be traced in sections into the region of the terminal nucleus of the vagus.

But most extraordinary of all, there was a group of fibres in two cases which curved downwards (caudally) in the neighbourhood of the clava and descended on the surface of the posterior columns.

The most instructive of these two cases is that of an Egyptian boy (æ. 2 ann.) shown semi-diagrammatically in Figs. 2, 3 and 4, representing the ventral, left lateral and dorsal aspects respectively of the medulla oblongata.

Immediately below the pons both the pyramid and the fasciculus circumolivaris were covered by the upper part of the nucleus arcuatus (Fig. 2): the circumolivary bundle then emerged on the surface as a prominent strand (4.5 mm. broad and almost 1 mm. thick) in front of (*i.e.* superficial to) the lower part of the nucleus arcuatus, which intervened between it and the rest of the pyramid. A few scattered fibres of the circumolivary bundle occupied a more mesial and subsequently a more caudal position (Figs. 2 and 3) than the compact chief mass. The thin layer of

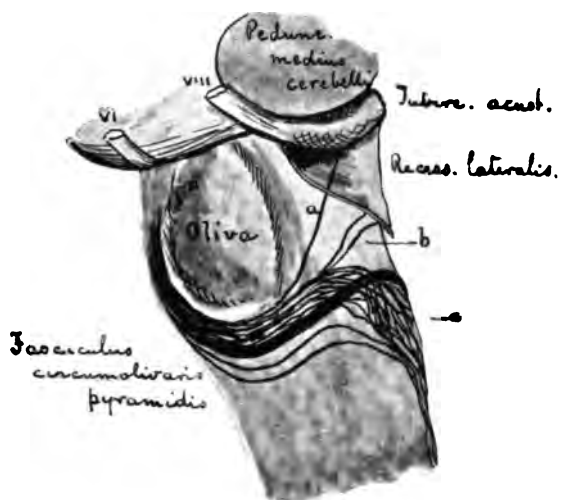


FIG. 3.

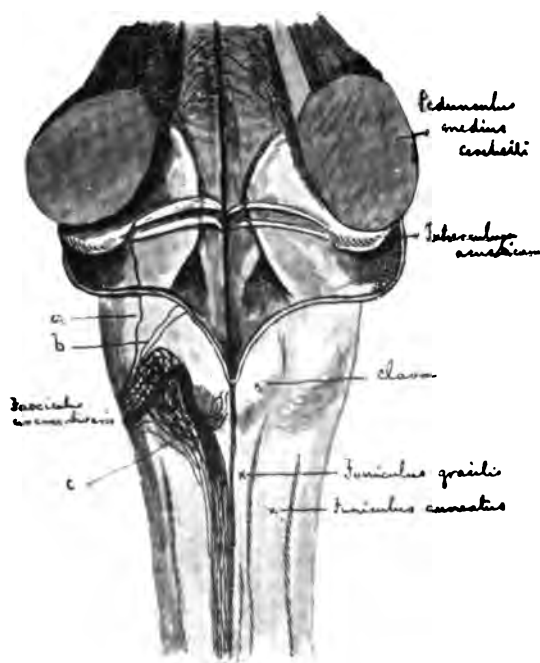


FIG. 4.



external arcuate fibres was placed on a more superficial plane than the aberrant pyramidal fibres (Fig. 2).

The circumolivary bundle then swept outward and backward around the caudal margin of the olive. On the lateral aspect of the medulla oblongata the compact strand became broken up into one large tract and numerous small scattered filaments. A small thread (Figs. 3 and 4, *a*) passed upward into the lateral recess of the fourth ventricle, where it disappeared from view in the tuberculum acusticum: a pair of small filaments (*b*) entered the floor of the fourth ventricle just below the inferior fovea: but the vast majority of the fibres passed backward to the upper end of the clava. The behaviour of this latter group was very peculiar. The large tract (which I mentioned above) passed backward with a slight upward obliquity until it reached the clava: it then suddenly bent downward (caudally), and after crossing the nucleus gracilis, entered Goll's column: it passed downward on the surface of this column and in the first segment of the spinal cord its fibres began to scatter, so that at the second cervical segment it became impossible to distinguish the aberrant pyramidal fibres from the other fibres of the postero-mesial column.

A group of scattered fibres of the circumolivary bundle, which were placed at first above the large tract (Fig. 3), bent downward near the clava and crossed the latter tract superficially (Figs. 3 and 4): many of these fibres appeared to terminate in the nucleus gracilis (or the grey substance in its neighbourhood), others accompanied the large tract into Goll's column. On the right side there was no circumolivary tract, but many of the pyramidal fibres were deflected from their course in an irregular manner on the surface and along the lateral border of the olive (Fig. 2 *x*). The olives were markedly asymmetrical, the left being much shorter and plumper than the right, as though it had been pulled upward by the sling-like circumolivary bundle.

Although fibres of the circumolivary bundle could be traced as far as the clava in most cases, in only one other instance was I able to certainly trace fibres of this bundle into the posterior columns. In that case, the tract was considerably smaller than that shown in Fig. 4.

In every one of my fifteen cases, as well as in that recorded by Barnes, the large circumolivary tract was on the left side. In

three of my cases, a very diminutive thread-like bundle of a similar nature was also present on the right side of bulbs, which exhibited left-sided tracts, ten or fifteen times as big.

It is not altogether clear why this aberrant grouping of the pyramidal fibres should show such a noteworthy preference for the left side. Perhaps there is a greater tendency for fibres to be "crowded out" of the pyramidal tract which comes from the hemisphere pre-eminently concerned with the more skilled movements.

It is difficult to offer any adequate explanation of the significance of this tract. Barnes states that it is possibly "an extra (uncrossed) source of pyramidal supply to the nuclei of the vagus and hypoglossal" (*op. cit.* p. 499).

The presence of descending fibres in the posterior columns coming from the pyramidal tract is, I believe, a new observation for the human brain. It is a well-known fact that *crossed* pyramidal fibres descend in the posterior columns of many rodents and ungulates (for the literature see Goldstein, *Anatomischer Anzeiger*, Feb. 20, 1904); but these fibres occupy the ventral corner and not the dorsal surface. These posterior column fibres presumably terminate among the cells of origin of the upper motor roots of the spinal nerves.

I have published this incomplete preliminary note in the hope that future investigators of degenerated pyramidal tracts may look out for this recurrent circumolivary bundle, and solve the problem of its ultimate destination. There can be little doubt as to the termination of some of the fibres in the nuclei hypoglossi et ambiguus and in the spinal cord. But the fate of the uppermost (ascending) fibres is very uncertain. Do they go to the nucleus facialis or do any of them enter the cerebellum?

*Postscript.*—During the two days that have elapsed since this note was written I have examined thirty more brains, and have found five more cases of this recurrent circumolivary bundle, four cases in Egyptian brains and one in a Syrian.

One of these (Egyptian) brains presented the unique feature of possessing very large recurrent tracts of approximately the same size on both sides of the bulb. In all the others (*i.e.* in twenty cases out of twenty-one, counting Barnes' specimen) the large bundle was present on the left side. In no case (of the whole twenty-two) was the right tract larger than the left.



In Stanley Barnes' abstract of a memoir by A. Pick in the first volume of this *Review* (p. 798), there is a reference to "pyramidal fibres which split off from the surface of the medullary pyramid, wind round the olive with the external arcuate fibres, and make their way to a position near the hypoglossal nucleus." Pick regards them as "fibres pyramidales homolaterales superficielles" of Madame Déjerine.

When my note was written I was not acquainted with this abstract or with Pick's memoir.

[Since this note, including the postscript, has been put into print, I have seen five more examples, making 25 in all, of this circumolivary bundle. One of these is of especial interest because it is situated on the *right side*.]

## Abstracts

### ANATOMY.

**THE CENTRAL CONNECTIONS OF DEITERS' NUCLEUS AND (138) ITS NEIGHBOURING NUCLEI.** VAN GEHUCHTEN, *Neuraze*, Vol. vi., 1904, p. 21.

THE experimental work for this paper was performed on rabbits, Marchi's method being used for the preparation of the material. As the result of a lesion destroying the auditory tubercle, the lateral part of the restiform body, the lower portion of Deiters' nucleus and the inner part of the inferior cerebellar peduncle, Van Gehuchten found four strands of fibres degenerated: those were, (1) the vestibulo-spinal tract; (2) the posterior longitudinal bundle; (3) fibres of the dorsal auditory path; (4) fibres passing from the dorsal part of the restiform body towards the lateral nucleus of the medulla. The first three of these tracts are dealt with in this paper.

1. *The vestibulo-spinal tract*.—Van Gehuchten refers fully to the literature of this tract, the origin of which has been variously placed in the cerebellum or in Deiters' nucleus; he points out the need to supplement Marchi's by Nissl's method in trying to determine the cells of origin of any strand of fibres. Van Gehuchten sums up his results by stating that this bundle is a purely descending (motor) strand, coming entirely from Deiters' nucleus and passing through the anterior columns of the spinal cord into

the sacral region ; he has not been able to trace the endings of its fibres into the anterior horns of grey matter, as described by Probst and by Fraser.

2. *The posterior longitudinal bundle.*—Van Gehuchten refers to the conflicting views as regards the functions of this bundle, it having been described as composed wholly of descending (motor) fibres by Kolliker, Cajal and others, and as consisting only of ascending (sensory) fibres by Held, Tschermak and others ; he points out that he himself has described it as formed of both ascending and descending fibres since 1896. His present work confirms Van Gehuchten in his view that this is a sensori-motor strand ; he agrees with Fraser that ascending fibres are only found in the pontine and mesencephalic part of the bundle and are entirely absent from the bulbar and spinal portion. The ascending fibres for the most part pass by the heterolateral bundle (*i.e.* are crossed), only a few going into the homolateral strand (*i.e.* are direct) ; most authors place the origin of these ascending fibres in Deiters' nucleus, but Van Gehuchten regards this view as needing confirmation, pointing out that it is impossible to avoid damage to Bechterew's nucleus and the lateral tubercle when destroying Deiters' nucleus. Van Gehuchten does not agree with the statement that the homolateral ascending fibres come from the sixth nucleus (Bruce, Fraser) ; he has found total chromatolysis of this nucleus after division of the sixth nerve, so regards all its cells as giving rise to root fibres of this nerve. These ascending fibres can be traced to the fourth and third nuclei, and some of the heterolateral ones even further into the region of the optic thalamus. Of the descending fibres, those in the mesencephalic course of the bundle come from the nucleus of the posterior commissure (Probst ; Van Gehuchten) ; these are reinforced by strands arising from the nuclei of the eighth nerve, and to these is added a third set of fibres (reticulo-spinal) coming from cells scattered in the reticular formation of the pons and bulb. All these fibres can be followed into the antero-lateral columns of the spinal cord. Van Gehuchten states that the only physiological conclusion justified by the anatomical facts known about the posterior longitudinal bundle is that it forms a path connecting the terminal nuclei of the eighth nerve with various motor cranial and spinal nerves, and so permits of reflex movements in response to impressions conveyed by the cochlear, or, more probably, the vestibular part of the eighth nerve.

3. *Central auditory path.*—Van Gehuchten's recent work confirms the results he has already published (*Nervaze*, vol. iv., 1903) with regard to these fibres. They arise from the ventral auditory nucleus and pass chiefly by the corpus trapezoides to the opposite side anterior to the olives ; a smaller strand (bundle of Held)

constitutes the dorsal auditory path and crosses in the raphe to lie posterior to the superior olive; both strands then turn upwards to enter the lateral fillet and end in relation to the nucleus of the lateral fillet and the base of the posterior corpus quadrigeminum. Contrary to the results obtained by Probst, Van Gehuchten never met with degeneration in the auditory striæ after a lesion restricted to the ventral auditory nucleus. HEWAT FRASER.

**ON THE PEDUNCULUS CORPORIS MAMILLARIS, THE GAN-  
(139) GLION TEGMENTI PROFUNDUM, AND THE DORSO-  
VENTRAL RAPHE-FIBRES OF THE TEGMENTUM.**  
HATSCHKE, *Arch. a. d. Neurolog. Institute a. d. Univ. Wien*  
(Obersteiner), H. x, 1903, p. 81.

HATSCHKE accepts the generally recognised origin of the pedunculus corporis mamillaris from the lateral nucleus of the latter, and then attempts to decide between the very contradictory descriptions of its course and termination. His conclusions are drawn from study of the brain of *Parameles*, one of the monotremata, in which the tract seems to be unusually well developed, and is easily studied, as its whole course is visible in a suitable sagittal section. He finds that it ends in a group of cells, the ganglion tegmenti profundum, which lies ventral to dorsal longitudinal bundles at a level caudal to the decussation of the superior cerebellar peduncles. It is very variable in size, largest apparently in the monotremata, small or absent in apes, and never present in man. Indeed, its size seems to be directly dependent on the degree of development of the olfactory system. When present, it lies intermediate between the nucleus reticularis tegmenti dorsalis and the nucleus centralissuperior medialis. Accordingly, the Gudden part of the tegmental bundles of the corpora mamillaria also end in it, and with this Hatschek agrees. He has not been able to make out any of its other connections, and cannot verify Ziehen's description of the relation to it of the crossed fibres of the lateral fillet.

Other tracts, though not related to this system, are also described in *parameles*. The dorsal peduncular fibres of Russell—*faisceau en crochet* of Thomas, from the contra-lateral half of the cerebellum to the quadrigeminal region, are well developed. There is also a minute description of that part of the tegmental tracts of the corpora mamillaria which enter the pons as parallel strands on either side of the raphe ("dorso-ventral fibres") under the name of *fasciculi paramediani*, which seem to be more developed in the monotremata than in mammals. H. thinks they also receive fibres from the ganglion interpedunculare.

The ganglion tegmenti profundum is regarded as a centre, analogous to the nucleus centralis and other mid-brain nuclei, of origin of descending reflex, olfactory tracts, and through its intermediation and the connection of Wallenberg's basal olfactory tract with the dorsal longitudinal bundle, the connections of the osmotic system come into line with those of the visual and auditory apparati.

GORDON HOLMES.

### THE BASAL OPTICUS ROOT AND TRACTUS PEDUNCULARIS

(140) **TRANSVERSUS.** O. MARBURG, *Arb. a. d. Neurolog. Institute a. d. Univ. Wien*, H. 10, 1903, p. 66.

MARBURG describes and discusses the relations of that bundle of fibres which is best known in its course on the lateral surface of the mid-brain as the tractus peduncularis transversus. From experimental work, it is known that in mammals it springs from the retinal cells, runs backwards in the optic nerves, partly decussating in the chiasma, and finally ends in a ganglion of the mid-brain lateral to the pedunculus corporis mamillaris. A tract with similar course is present in amphibians, reptiles and birds, known as the basal opticus root, and in these classes ends in a relatively large and well-defined nucleus, the ganglion ectomamillare. No definite homologue has been recognised in fishes.

In addition to definite experimental evidence, the absence of the tract in amblyopic mammals, as the mole or blind mouse (*Spalax*) and in blind amphibians (*Proteus anguineus*), is an argument in favour of its origin from the eyes.

Marburg homologises the nucleus in which Kölliker and Bechterew saw the transverse peduncular tract end with the ganglion ectomamillare of the lower classes.

Besides fibres derived from the eye, the tract also probably contains some that come from the grey matter of the base of the brain and from the nucleus suprageniculatus.

GORDON HOLMES.

### EXPERIMENTAL RESEARCH ON THE COURSE OF THE OPTIC

(141) **NERVE FIBRES.** GEORGE DEAN and C. H. USHER, *Brain*, Vol. xxvi. No. 104, p. 524.

THE writers give a very interesting and valuable account of a series of experiments performed on monkeys, with a view to the determination of the position and course of nerve fibres in the optic nerve and in the chiasma. Degeneration was produced either by

division of the optic nerve or by a lesion of the retina. The method of procedure was the same in these experiments as in a former series published in the *Transactions of the Ophthalmological Society of the U. K.*, vol. xvi. In the former series rabbits were used, with one exception. In the present series monkeys were used throughout. For the methods of hardening, staining, etc., readers are referred to the former paper.

The cases dealt with in the present communication are divided into three groups according to the site of the lesion produced.

1. Division of the optic nerve.
2. Retinal lesions at a distance from the macula.
3. Retinal lesions at the macula.

In group 1 the optic nerve was divided in the orbit, with as little injury to blood-vessels as possible. Five monkeys were used, all *Macacus Rhesus*. The object in view in the performance of the experiments was chiefly to determine the difference in character and extent of degeneration in the parts of the nerve above and below the scar.

In three of the cases the degeneration as indicated by osmic acid was most marked above the scar. In these cases, however, there were profound changes below the scar involving largely the connection tissue framework. Osmic acid staining was much less marked below than above the scar. The changes found below the scar were regarded as largely due to interference with the vascular supply.

Results obtained in the chiasma:—

(1) The degenerated fibres begin to cross over as soon as they enter the chiasma.

(2) The crossing is first seen to take place at the ventral periphery, where the first junction of the nerves takes place. As the degenerated fibres are followed backwards they are found to occupy more and more of the chiasma from the ventral to the dorsal surface, till ultimately they are distributed over the whole central part.

(3) Only a few if any of the crossed fibres appear to approach the lateral periphery of the half of the chiasma corresponding to the unoperated nerve, whereas on the corresponding area of the opposite side the degeneration is dense.

(4) It could not be determined that the crossed fibres at the posterior part of the chiasma were more numerous than the uncrossed.

(5) A very definite tract of crossed fibres lies at the central part of the chiasma posteriorly.

In group 2 are four cases of lesion at a distance from the macula, three in *Macacus Rhesus* and one (case 9) in the Bonnet Monkey. In all these the degeneration occupies a position in the

sectional area of the nerve corresponding to the site of lesion in the retina.

Results in the chiasma :—

(1) A ventral degeneration in the nerve maintains its ventral position in the chiasma. A very few degenerated fibres cross to the other side.

(2) A lesion at the inner part of the fundus is followed by a degeneration of the inner part of the nerve which in the front part of the chiasma lies on the same side, but ultimately crosses completely to the opposite side.

(3) A very peripheral lesion in the outer part of the fundus is followed by a degeneration in the outer dorsal part of the nerve. In the chiasma the degeneration lies on the same side close to the lateral periphery, ultimately crossing and occupying the ventral periphery of the opposite side. Only a few fibres remain uncrossed at the posterior part of the chiasma. The course of the degeneration throughout the chiasma in this instance was attributed to the fact that a specimen of the Bonnet Monkey was employed, and it was thought probable that the more primitive type may have obtained in this instance.

It is, however, noted that another observer (Parsons) has found degenerated crossed fibres in the chiasma following upon a temporal lesion of the retina both in the Bonnet Monkey and in *Macacus Rhesus*.

In group 3 retinal lesions in the macular region were made by means of a fine thermo-cautery in three monkeys. In a fourth, in which the ophthalmoscopic appearances of the outer parts of the optic discs were those of a restricted atrophy, no experimental lesion was performed.

In all four the tissues were subjected to microscopic examination.

In the optic nerve the degeneration was found in all four cases to occupy a position at the outer side in front. Farther back in the nerve the degeneration was central, and this position was maintained to the chiasma.

In the chiasma the macular fibres occupy a central position at the anterior part on each side, but soon pass towards the dorsum. They occupy a dorsal position before crossing begins, and the first evidence of crossing is seen at the dorsal periphery, some distance behind the front of the chiasma. Farther back the macular fibres tend to spread from the dorsal position towards the ventral aspect, though they never reach the ventral periphery.

The writers of the paper are unable from their evidence to say definitely whether or not all the macular fibres cross at the chiasma.

Vossius and Bunge found that the macular fibres in man

occupied a dorsal position in the chiasma, while Henschen believed the position of these fibres to be central throughout the nerve, chiasma and tract. This paper is illustrated by excellent plates showing microscopic appearances, with a diagrammatic scheme showing the position of the optic nerve fibres in the chiasma.

A. H. H. SINCLAIR.

**ON THE LIGHT PIGMENT IN THE NERVE CELLS AND THE  
(142) OCCURRENCE OF FAT-LIKE BODIES IN THE CENTRAL  
NERVOUS SYSTEM. OBERSTEINER, *Arch. a. d. Neurol. Instit.*  
*a. d. Wiener Univ.*, H. 10, 1903, p. 245.**

ACCORDING to this author the nerve cells of the new-born are free from pigment. It is acquired in later years and increases in amount with advancing age. There are two kinds of pigment, light and dark. They differ from each other chemically, and also in their distribution. The light variety is closely allied to fat.

Obersteiner has studied the light pigment in the entire nervous system of two cases, one aged sixty-five, the other aged eighty-five. Marchi's method was employed to demonstrate the granules. His conclusions are as follows:—

1. There are lipophobe cells which even in advanced life remain free from fat pigment. In senility the cells may contain a small number of very fine granules, *e.g.* Purkinje's cells and those of the Edinger-Westphal nucleus.

2. Lipophile cells which contain a considerable quantity of pigment. These are divided into (*a*) cells in which the pigment is heaped into a thick mass, while the remainder of the cell body is to a greater or less extent free, *e.g.* anterior horn cells, pyramidal cells; (*b*) cells in which the granules are distributed more equally and less thickly throughout the protoplasm, *e.g.* Clarke's column, the inferior olive. Most small cells belong to this type. Large cells are for the most part richest in pigment.

As to the origin of the granules, Obersteiner is of opinion that they are formed at the expense of some constituent of the cell which is not the conducting substance. In how far the Nissl bodies provide the material for their formation the author is not prepared to say, and in this connection he thinks it worthy of notice that similar formations occur in the glia cells. Further, in the anterior horn cells the pigment is found heaped in one spot, while in the remainder of the cell the Nissl bodies are normal and between them no fat granules are to be seen.

In order to explain why only some cells possess pigment, Obersteiner adopts the theory that it is a product of some involutive or regressive change which appears in the various cell types at

different times, *e.g.* earliest in the anterior horn cells and latest in Purkinje's cells. It is probable that the latter possess more resistive power than the former.

Pigment granules exist in the white matter of the cord. In a senile brain, individual granules were found, but in the cortex, never arranged in rows parallel to the surface, indicating degeneration of the tangential system.

Pigment occurs in many glia cells. It increases in amount with age, and its chemical reactions are similar to those of nerve cell pigment. In senile brains especially, the glia cells also contain clear refractile bodies with a darkly-stained margin. Pigmented glia cells in the cortex are found mostly in its outer third.

In the molecular layer of the cerebellum Marchi's method gives no reaction, and it is doubtful if glia cells exist in this situation. In the granular layer there are a few scattered pigmented glia cells, and also in the white matter. In the cord the glia cells are not rich in pigment. Those containing fat are most numerous in the vicinity of the posterior roots.

In the choroid plexus there are yellow-brown granules which stain black with osmic acid. These are not present in the new-born. The ependymal cells show a similar reaction.

In the adventitia of the vessels there is always fat pigment both in the new-born and in the adult. It is a yellow-brown pigment which with osmic acid turns light grey. DAVID ORR.

**CONTRIBUTION TO THE STUDY OF THE ENDOCELLULAR  
(143) NETWORK IN THE NERVE ELEMENTS OF THE SPINAL  
GANGLIA.** SOUKHANOFF, *Névrose*, Vol. vi. f. 1, p. 75.

In order to demonstrate the endocellular network in the cells, the author uses the method of Kopsch, which is as follows:—Place the fresh ganglia in 2 per cent. osmic acid, and leave in the dark for three weeks. They are then washed rapidly in water, dehydrated rapidly by a few hours' immersion in alcohol, and embedded in paraffin.

Soukhanoff has observed an intracellular reticulum in the nerve cells, greatly resembling that of Golgi. It consists of filaments, curved and freely anastomosing, with swellings at the nodal points. Some of these are fine, others of larger calibre. The endocellular apparatus does not reach the periphery of the cell, but is surrounded by a clear protoplasmic layer. It has no immediate connection with the nucleus, does not penetrate it, and is therefore perinuclear. The author is of opinion that since Golgi and Kopsch get an identical appearance by two different methods, this network cannot be an artificial product.

DAVID ORR.



**A CONTRIBUTION TO THE ANATOMICAL STUDY OF THE  
(144) POSTERIOR COLUMNS.** J. NAGEOTTE, *Nouv. Icon. de la  
Salpêtrière*, jan.-fév. 1904, p. 17.

THIS important paper is based upon the investigation of a case of tumour compressing the sacral and lower two lumbar roots in the cauda equina, and of a case of incipient tabes dorsalis in a general paralytic. In using the term "incipient tabes," the author wishes to denote a condition in which only certain fibres of the posterior roots forming a distinct system have been affected by the morbid process; in this instance the degenerated fibres were limited to the region known as the external bandelette of Pierret in their intramedullary course. The cases were examined by the Marchi, Nissl, hæmatoxylin, carmine and Weigert-Pal methods, but the last-named was the most important from the anatomical point of view, owing to the length of duration of the lesions. The conclusions arrived at are the following:—

- (1) The endogenous fibres of the posterior columns of the lumbo-sacral region may be divided into large and fine fibres.
- (2) The large endogenous fibres form (a) a major part of the cornu-commissural zone, and (b) the median sacral triangle of Gombault and Philippe. The latter represents the inferior extremity of the bundle of Hoche found in the dorsal region of the cord.
- (3) The median triangle is distinct from the oval area of Flechsig, which is of exogenous origin.
- (4) The fine endogenous fibres are in part horizontal and in part vertical; the latter are scattered diffusely in the column of Burdach; a few are found in the column of Goll in the cervical region.
- (5) Lissauer's zone is composed of fine vertical fibres of endogenous origin; it degenerates late in tabes and is not degenerated in pure root lesions.
- (6) The net-work of fine fibres in the posterior horn is of endogenous origin.
- (7) Clarke's column does not receive fibres from posterior roots lower than the 3rd lumbar.
- (8) The external bandelette of Pierret is never in contact with the posterior horn, and in the lower dorsal region presents a complicated shape something like an M on each side of the median septum.
- (9) The external bandelette contains radicular fibres of medium length which remain within its limits throughout their intramedullary course; those of the lumbo-sacral region do not reach the column of Goll.

- (10) The long radicular fibres do not enter the external bandelette, but pass through the postero-external fields.  
 (11) The marginal zone of Westphal contains only short radicular fibres in addition to endogenous fibres.

Some of the above results are important contributions to our knowledge of the anatomy of the posterior columns; others are not entirely confirmed by the study of cases in which the lesions have been sufficiently recent to allow of investigation by the Marchi method.

E. FARQUHAR BUZZARD.

#### THE CEREBRAL CONNECTIONS OF THE OCULAR MOVE-

(145) MENTS. BERNHEIMER, *Von Graefe's Arch. f. Ophthalm.*, lvii. 2.

It is well known that the angular gyrus must have some relation to ocular movements, for stimulation of it, and more particularly of certain areas in it, is productive of movement of the eyes. By very carefully applying a mild stimulus these movements can be set up without other complicating actions, such as turning of the head, and in the monkey the eyes may be directed upwards, to the right, etc., at the will of the operator. A certain bundle of fibres, which runs from the large ganglion cells of the visual area to end in the nuclei of the eye muscles, brings about a close relationship between the visual and the oculo-motor regions, but this is not to be regarded as the sole, or even as the chief, path. It is too small; besides, when it is stimulated mildly, nothing results, and it is only when a fairly vigorous stimulus is given that binocular movements take place. In a few apes Bernheimer has succeeded in removing the corpora quadrigemina and yet keeping the "patient" alive for a time. He found that movements caused by stimulation of the visual area then took place no less readily or correctly than when the corpora quadrigemina were present. In another series of cases he cut through all the tissues underlying the aqueduct, even so far as through the nuclei of the third and fourth nerve nuclei, keeping his incision in the precise middle line. In certain of the cases destruction of the cortex of the corpora was combined with this section, but in neither case did the same strength of current suffice to bring about synergic eye movements. Bernheimer urges, then, that the corpora quadrigemina must not be regarded as forming a reflex centre for ocular movements.

He next removed the entire left angular gyrus in three monkeys, of which two were subsequently in a fit state to enable him to make investigations. He found that after this procedure the head could be moved either to right or to left, but that, without the head, the eyes were incapable of movement to the right. He then studied carefully on the hardened brain the path of

degeneration following such section. After leaving the cortex the fibres rapidly converge to form a somewhat flattened bundle; they then spread out again as they are distributed in the substance of the optic thalamus.

W. G. SYM.

## PHYSIOLOGY.

**ON THE DILATATION REFLEX OF THE PUPIL TO DIMINISHED ILLUMINATION.** SIEGMUND KREUZFUCHS, *Arb. a. d. Neurolog. Institut. a. d. Wiener Univ.*, H. x., 1903, p. 275.

THE writer supposes that on shading the eye or reducing the illumination, a nerve stimulus is started in the eye, conveyed to the dilatation centre, and thence back to the iris, causing dilatation of the pupil. What, then, is the course of these centripetal fibres? Are they in the optic nerve or in the trigeminal? Section or atrophy of the optic nerve is followed by dilatation of the pupil. We therefore suppose that the centripetal fibres dealing with reflex contraction of the pupil run in the optic nerve, while those causing dilatation do not, and, therefore, after section of the nerve, the dilatation reflex is free to act alone. The motor paths for the dilatation reflex have been shown by Budge and others to be in the sympathetic, the fibres coming from the 7th and 8th cervical, and 1st and 2nd dorsal nerves. Budge placed the centre for dilatation between the 6th cervical and 3rd dorsal vertebrae, but supposed that a centre situated at a higher level must also exist. Budge also found that section of the trigeminal nerve caused contraction of the pupil, especially if the section were placed external to the Gasserian ganglion. Other observers have confirmed this observation, but the explanations given have differed widely. Mr Kreuzfuchs experimented on the rabbit by cutting the trigeminal near the base of the brain. Immediately after section of the nerve the pupil on that side contracted, but this contraction diminished after a time. He noted, however, that while in ordinary daylight the pupils might appear of equal size, in a darkened room the pupil on the side of the intact nerve dilated widely, while on the side where the nerve had been divided it remained of medium size. That the motor dilator fibres were not divided was shown by the fact that exposure and stimulation of the cervical sympathetic caused maximal dilatation of the pupil. The contraction of the pupil following section of the 5th nerve must, in the opinion of Kreuzfuchs, be due to section of centripetal fibres concerned with reflex dilatation. Kreuzfuchs also examined three cases in which the Gasserian ganglion had been destroyed in a surgical clinic. In the first case the pupils were equal in bright daylight, and reacted equally. In a dimmer

light the pupil on the operated side appeared smaller, and reacted less actively. In the second case the pupils did not react to light, and in the third case no difference in the pupillary reactions could be detected. Krause has paid attention to the behaviour of the pupils in man after operations on the Gasserian ganglion, and has found no evidence of any noteworthy change.

From his observations on the rabbit and in man, Kreuzfuchs considers that the fifth nerve certainly contains centripetal fibres for dilatation of the pupil in a dim light. He contests the view of Bechterew and others that dilatation of the pupil is due merely to an inhibition of the contraction centre in the 3rd nucleus. The Robertson phenomenon, he supposes due to an interruption of the centripetal impulses to the dilatation centre in the spinal cord. The myosis so often associated with the Argyll-Robertson pupil might thus be due to the unopposed action of the sphincter.

An extensive bibliography is appended to the article.

J. V. PATERSON.

**RESPIRATORY MOVEMENTS OF THE GLOTTIS IN THE**  
(147) **RABBIT.** F. DE BEULE, *Nervaze*, Vol. vi., 1904, p. 3.

THIS paper is a continuation of one recently published on respiratory movements in the dog (*Nervaze*, vol. iv. fasc. 1, 1902, ref. *Rev. Neur. et Psychiat.*, vol. i. p. 746). The general results are very similar to those already noted: during quiet respiration in the rabbit, as in the dog, the glottis remains freely open, either motionless or showing slight oscillations; during forcible (frightened) respiration the rima enlarges with each inspiration; the onset of asphyxia is marked by spasmodic closure of the glottis; the cadaveric position shows a much smaller opening of the glottis than that found in the living animal, indicating a constant tonic activity of the abductors during life. Beule notes two points of difference between the dog and the rabbit; in the latter the glottis not only enlarges transversely with inspiration but also antero-posteriorly, owing to the arytenoids rocking backwards with the cricoid cartilage as the result of the contraction of the cricothyroid muscle; the relatively larger size of the glottis in the dog explains the absence of this antero-posterior movement. The second difference lies in the wide opening of the glottis with forced inspiration being unattended by expiratory contraction in the rabbit; the abductors act forcibly, but the adductors remain quiescent. In the rabbit, as in the dog, the glottis still shows respiratory movements after all the laryngeal muscles have been paralysed; the movement of closure is due in both animals to the contraction of the constrictors of the pharynx which compress the

alæ of the thyroid cartilage and so bring the arytenoids together in this way approximating the vocal cords. The movement of opening, due in the dog to the stylopharyngeus muscle, is differently effected in the rabbit; here it is brought about by the contraction of the muscles of the tongue, which, pulling on the hyoid bone, cause traction on the mucous membrane reflected over the arytenoid cartilages so that these are slightly separated. Division of the hypoglossal nerves at once stops this indirect abduction of the cords.

HEWAT FRASER.

**NERVOUS FATIGUE AND THE ENERGY OF CONTRACTION IN  
(148) VOLUNTARY MUSCULAR WORK.** Z. TREVES, *Arch. di  
Fisiol.*, Florence, Jan. 1904, p. 170.

THE writer discusses the question whether the nervous energy which excites muscular contraction is subject to the laws of fatigue or not, and to what extent its manifestations can be studied. He has shown that when one is studying fatigue by work with a moderate weight the curve obtained descends rapidly to a constant level, at which the work can be maintained practically indefinitely. That is to say, a point is reached after which muscular fatigue does not increase. But the question then arises as to the effect of such continued work upon the energy of the nervous centres. Does the available nervous energy continue constant, or is there another form of fatigue which is not manifested by the muscular contractions?

This is a complicated question, and as a contribution to the solution of the problems involved the author has studied the variations in the *energy of contraction* during voluntary muscular work. He shows that the energy of contraction bears no sort of relationship to the curve representing work done. As the ordinary form of the ergograph represents the latter only, the author has had to make use of a modification of that instrument for his researches.

The instrument made use of is provided with a metal wheel which revolves during the contraction of the muscle, and to which a certain momentum is given. The more quickly and energetically the movement is made, the greater the momentum given to the wheel, which continues to revolve after the muscular contraction has ceased. The amount of the revolution so obtained can be read off on a scale, and is taken as indicating the energy of that particular contraction.

The experiments described refer to two points—the influence of the weight and the influence of the rhythm upon the energy of contraction.

As to the former, the experiments show that there is an optimum weight, with which the mean energy of contraction is greatest. With weights either lighter or heavier than the optimum, the mean energy of contraction is less. This optimum weight is found to be the greatest weight to which the subject can impart a velocity greater than that of the finger. In the experiments the optimum weight varied between 4 and 6 kilogrammes.

With regard to rhythm, the study of the mean energy of contraction proves that rhythm constitutes an essential cause of fatigue, whether it is determined by a metronome or left to the choice of the worker.

In order that the work may proceed at a constant level, it is necessary that the rhythm should not surpass a certain frequency, which is found to bear a relation to the intensity of the work; that is to say, to the weights to be lifted. In many industrial occupations the conditions necessary for the production of a maximum of work with a minimum of fatigue are not realised. For instance, in supplying a machine with material, the actual exertion involved in each movement may be slight, but the rhythm is too frequent, and the worker is therefore in a condition highly favourable to the production of fatigue, but fatigue of a kind which would not be shown in a curve in the production of work. This is the indefinite kind of fatigue which is felt at the end of a day's work, and whose seat is doubtless in the nervous centres. Its great importance lies in the fact that when we are trying to produce a maximum of useful effect in a minimum of time, our intelligence and will may carry us on until we are insensibly brought into a condition of nervous exhaustion—a conclusion which coincides with the teaching of the ordinary facts of life, but not very well with the opinion widely accepted among physiologists, that fatigue exercises a protective function in the organism, whether it be that the nervous system protects the muscles at its own expense, or that the muscle preserves from exhaustion the psycho-motor functions by a peripheral paralysis.

W. B. DRUMMOND.

**ACTION OF RADIUM ON THE EXCITABILITY OF THE  
(149) CEREBRAL CORTEX.** GIULIO OBICI, *Riv. di Patolog. nerv.  
e ment.* February 1904, p. 76.

DR OBICI has been engaged for some time in investigating the action of radium on the functions and structure of the nervous system; in this communication he describes a series of experiments on eels, devised to test the effect of radium on cortical excitability with the faradic current.

The method of experiment was as follows:—Slits were made in

the cranium of the animal on either or both sides of the median line, after which the eel was left to rest for from thirty minutes to three hours. The faradic current was then applied, and the minimum necessary to produce the contraction of a given muscular group noted. The emanations of a gramme of bromide of radium and barium were then allowed to play on the cortex through the slit for ten, twenty or thirty minutes, after which the current was again applied, and the minimum required to give the same contraction as before noted. As a control experiment the current was applied at the same intervals to the second hemisphere of the brain, which was, however, sheltered by a steel plate from the radium emanations.

Dr Obici's results were:—

1. There was a constant increase in the electrical excitability of the cortex after ten, twenty or twenty-five minutes' application of the radium rays. The increase of distance between the coils of the battery varied from .5 to 3 centimetres. Sometimes violent muscular action would break out simply under the influence of the radium, without the application of the current. The control never showed an increase of excitability, but often a diminution.

2. The effect of the radium showed itself by increased cortical excitability only after about ten minutes when the *dura mater* was cut, and fifteen when it was left entire.

3. If the radiation is continued for about an hour, the excitability being tested by the current every ten or fifteen minutes, there is an increase only during the first thirty to forty minutes, and thereafter a decrease; unless the *dura mater* be left intact, in which case the increase may continue throughout the hour.

4. The increase of cortical excitability lasted from half an hour to three hours, and was followed by a return to the normal, or more frequently by a depression.

In all cases the radium produced a notable hyperæmia of the *pia mater* and of the cortex. After it had been applied for ten to twenty minutes, the light grey colour of the nervous substance had disappeared, giving place, first to a bright, then to a dark red. The *pia mater* becomes cedematous and turgid, and the cortex veiled by an exudation, resulting either from small hæmorrhages or from diapedesis from the dilated vessels.

These last facts are certainly of great importance for the explanation of the variations in cortical excitability. The slight hyperæmia of the first minutes, by bringing a better blood supply to the cortex, stimulates it, and lowers the threshold of its excitability; but when the hyperæmia becomes so intense as to bring about small hæmorrhages or exudation of the elements of the blood, then there succeed phenomena of an opposite nature—depression and paralysis.

If it is once admitted that the modifications of cortical excitability are connected with the vaso-motor disturbances, then the variations observed to follow upon even short periods of rest are also easily explained.

MARGARET DRUMMOND.

## PSYCHOLOGY.

**PSYCHOLOGICAL AND PSYCHIATRIC CONTRIBUTIONS TO**  
(150) **THE STUDY OF THE EGO.** *Psykiat.-psycholog. studier till jagets mekanism.* Frey Svenson, Stockholm, 1903.

THE author wishes to give an insight into the "mechanism" of the individuality from the standpoint of natural science. He refrains from dealing with any transcendental matter, and does not even hold a parallelismus theory to be necessary. As an introduction, he gives a review of the opinions of the relation between mind and body and of the views of the ego, especially met with in German authors of the nineteenth century.

The ego is defined as the bodily and mental structure, peculiar to each individual, as far as expressed in consciousness. The central constituent part in the ego is the *idea of the body* (Koerpervorstellung), composed of sensations from external sense-organs and from internal organs. In its totality the idea of the body is rarely present in the consciousness, but penetrates into it in every moment of failing attention. When an idea is to be reproduced, the organ-sensations do not amalgamate with other sensations at the same time, and it is due to this discontinuity that an idea can be distinguished from a hallucination. According to the theory of the author a hallucination occurs when an organ-sensation by a morbid process tends to become reproduced together with the rest of the content of perception. A hallucination in the proper sense results, when the ideas are "autochthon" (Wernicke); if not, we have what is called the phenomenon of hearing of ideas (Gedankenlautwerden). As representing the actuality of the body, the organ-sensations make it possible for us to distinguish between body and mind and between an external world and an internal. They would not, however, give us our complete apprehension of the body if they were not brought into connection, in consciousness, with feeling. Feeling represents in consciousness the instincts (using the latter as bodily states, which are determined by the tendencies of the organism and which regulate our actions).

The favourable or unfavourable relation of the cells of the organism to the instinct of self-preservation is expressed in the consciousness by the most important of all feelings, the general



*feeling of the body* (coenaesthesia). This coenaesthesia is the foundation of what the author calls the *general emotional state*, which is the sum of all present feelings. The most essential and constant feelings are "somatic"; they are modifications of the coenaesthesia, such as hunger, thirst, fatigue, sexual desire. Its distinctive feature, however, the general emotional state gets from "mental" feelings, which stand in closer relation to ideas originated from the external world, such as grief, joy, anger, etc.

Against Lehman the author maintains that feelings exist without any intellectual motive. It must be remembered, however, that Lehman differentiates between normal emotions and those produced by intoxication or by diseases of the nervous system; the latter emotions are admitted to be without logical foundation. Feeling is a process quite as independent as the intellectual processes are. In feeling a subjective element is present, not due to organ-sensations of extracerebral origin, which subjective element is most probably the expression in consciousness of the vascular conditions of the cerebral cortex. Accompanying an idea this element appears as its "feeling-accent" (Gefühlsbetonung); more intensified, it forms the emotions; if the cerebral vasomotor state, as expressed in consciousness, is of some duration and connected with vasomotor and other changes in other organs, an emotional state (Stimmung) is produced. In the last resort the feelings depend on the condition of all the cells of the organism, even though the brain cells play the greatest rôle. Instincts and feelings based upon psychical motives join with the "feeling of the body," which is the chief factor in the general emotional state, and this general emotional state forms the basis of our individuality.

In the study of intellectual phenomena the author departs from the aphasia-scheme of Wernicke; as an objection to its psychology he instances its underestimate of the importance of feeling. From a naturalistic point of view the intellectual processes are reduced in significance to being merely servants of the instincts. Ideas *per se* do not possess any defined "feeling-accent," but acquire such through association with feelings of any kind. The "accent" is therefore not only that of like or dislike, but even that of hunger, fatigue, love, etc. The power of ideas to call forth feelings depends upon the relation of their content to the general emotional state. On the other hand a feeling gives to the ideas associated with it their *tension*, i.e. their tendency to appear in consciousness with any emotion which includes the feeling in question. Since further the supremacy of any idea serving a definite purpose is due to some craving, which in its turn depends upon feeling, the general emotional state becomes the determining factor in intentional thinking. But speaking generally, the

sum of all such ideas represents to the individual the idea of his mental ego. This idea is therefore constituted by the general emotional state, which ultimately is determined by all tendencies immanent in the cells of the body. In this way the idea of the ego is the expression of the individual organism.

In the second part of his work the author tries to prove that in the case of the insane, changes in the ego are preceded by a primary change in the emotional state, and that every change in the latter leads to changes in the personality, even though this transformation of the ego may not be always directly present to the consciousness (*e.g.* in manio-depressive insanity). In almost all cases of insanity therefore the ego is changed. If the memory is damaged, the content of the idea of the ego is diminished (*e.g.* in general paralysis, secondary dementia, later stages of dementia præcox). If the intentional thinking is disordered, the content of the idea of the ego lacks the combination and definiteness required for its completeness (*e.g.* in confusional states, earlier stages of dementia præcox). In one of the author's cases, an individual suffering from hypochondriac depression, and insensible to external affective impressions, whose memory and intellectual powers, however, were undamaged, referred to himself as "he," and later on as "it" or "the bone." There was in this case, according to the author, a pathological alteration in the general "feeling of the body." In such a case there is ability to reproduce intellectual, but not emotional processes. The syndrome of *negation* on the contrary presupposes the possibility of a comparison between present depression and a previous happier emotional state, and in its typical form it is characterised by the exaggeration of pleasant memories (*délire de satisfaction*). As a rule the negative ideas concern only the individual and are not generalised, except when associated in certain cases with fright and deficient orientation.

In cases of *possession* (demonopathia) usually there is no longer one undivided personality, though there need not be an interruption of the ego. The condition for this symptom is instability of the emotions, which oscillate above and below the normal. As the representative of the pathological and more constantly occurring state of emotion, a "parasitic ego" is created; on account of abnormal organ-sensations and verbal motor hallucinations, the patient places it inside himself. The emotional state is that of religious depression. As pseudo-demonopathies the author describes cases with a constant state of depression, in which the ideas of possession merely seem to be a symbolic expression of feeling of sinfulness.

Insanity of *persecution* (paranoia) is neither exclusively nor mainly a disorder of the intellect. In typical cases all intellectual processes go on in a normal way. Where, however, the paranoiac's

own person is concerned, the postulates of the normal individual for a causal nexus are abandoned. Here too the emotional state is the basis of wrong ideas. The feelings pathologically affected belong to the group of feeling of relation, *i.e.* the feeling of personal value and of position of dependence. In proportion as environments act for or against the instinct of self-preservation, the feelings of relation are favouring or adverse. It will thus easily be understood, that almost all false ideas can be simply classified as follows: ideas of inferiority or of grandeur, of persecution or of support. As a special group (acute hallucinosis) Wernicke has distinguished cases in which there is a rapid systematisation of wrong ideas and in which further ideas have the tendency to be reproduced with the distinctness of a perception, in other words to take the form of hallucinations. For the genesis of ideas of persecution and grandeur, however, these peculiarities are of little importance; the content of the ideas is determined in the same way in the group just mentioned as in others.

HANS EVENSEN.

## **PATHOLOGY.**

### **ON CYTODIAGNOSIS IN NERVOUS DISEASES. C. L. DANA.**

(151) With a description of *The Technique of Cell-Diagnosis*. F. W. HASTINGS, *Med. Rec.*, Jan. 23, 1904.

AFTER a short résumé of our knowledge with regard to the cerebro-spinal fluid and some remarks on the technique of lumbar puncture, Dana gives the results of his examination of twenty-two cases. He used Widal's technique, but after centrifuging counted the corpuscles without fixing and staining. This total enumeration is held by Widal to be no more accurate than his own method of estimating the degree of lymphocytosis on a dried and stained preparation. Dana's results agree generally with those of other observers: in eight cases of general paralysis there was a marked lymphocytosis; in four other cases, with absence of lymphocytes, the technique was not sufficiently rigorous. In no case of alcoholic psychosis was there a lymphocytosis. In a case of confusional psychosis following on injury, lumbar puncture, performed five weeks after the operation, disclosed a large number of red blood corpuscles, thus making almost certain that there had been some laceration and hæmorrhage, and that the psychosis was not merely due to mental confusion after concussion. The headache which sometimes follows lumbar puncture is occasionally reflex or psychical, according to Dana, for in one case where no fluid was obtained the headache came on.

Hastings, in his account of the technique, calls attention to the fact that wet preparations give fewer cells in a field than dried and stained preparations.

C. MACFIE CAMPBELL

**LYMPHOOCYTOSIS OF THE CEREBRO-SPINAL FLUID.** J. FRAEN-  
(152) KEL, *Med. Rec.*, Jan. 23, 1904.

FRAENKEL gives the results of the cytological examination of the cerebro-spinal fluid of thirty-three cases. In the seven cases of tabes examined there was a marked lymphocytosis; in one case of general paralysis out of four no lymphocytes were found, in the other three the lymphocytosis was marked. In six out of seven cases of multiple sclerosis there was a marked cellular reaction, but no polynuclears. In conclusion, Fraenkel says, "a positive inference cannot be made, as far as my investigations go, from the prevalence of one or other type of corpuscle." This is not the experience of most of those who have worked at the subject.

C. MACFIE CAMPBELL

**PRACTICAL DEDUCTIONS FROM THE REFUTATION OF THE**  
(153) **NEURONE THEORY.** DEBRAY, *Journ. de Neurolog.*, mars 20,  
1904, p. 101.

THE author only touches on the arguments which, in his opinion, have led to the refutation of the neurone theory, these being chiefly the recent anatomical work of Apáthy, Bethe and others, the retrograde degeneration of Nissl, and the fact of autogenous regeneration of the peripheral ends of sectioned nerves. To these he adds a few indirect clinical arguments of relatively less weight and value.

Accepting this refutation, his paper deals with the question from a clinical point of view. If there be definite anatomical continuity of cell with cell through the central and peripheral anastomosis of the fibrils, the fibrils of any axis-cylinder do not all come from the same source, and there is presumably also physiological continuity, *i.e.* the fibrils from these various sources may in their course innervate the various organs they come into connection with, and the one fibril is not more concerned than any other in any function, so that on the death or destruction of one its neighbour may replace it.

If the function of a peripheral organ be the best stimulant to the corresponding nervous centre, he argues that if there be, as he assumes there are, multiple connections between that organ and its centre, the integrity of the latter or its regeneration, if part of

it has undergone change, is best effected by local stimulation, as massage, passive movements and electrical treatment of the peripheral organ be muscle.

Cells which have undergone change as the result of section of their axis-cylinders can again become functional by sending out impulse through their secondary connections, and need only degenerate when these too are destroyed, as when the nerve is torn out. The effect of the re-education of movements is also explicable in the doctrine of continuity. Attention is also drawn to the possible regeneration of nerves from their segmental neuroblasts, and to the probability that local treatment of the peripheral organs may stimulate this regeneration. In this way local treatment may act in neuritis when only some portions of the fibres are affected.

It is remarked that the prognosis in nervous lesions is less gloomy on the assumption of the doctrine of continuity.

GORDON HOLMES.

**THE PATHOLOGY OF CHRONIC ALCOHOLISM.** W. FORD  
(154) ROBERTSON, *Brit. Journ. Inebriety*, Vol. i. No. 4, April 1904,  
p. 226.

In this paper Dr Ford Robertson deals with the action of alcohol on the individual and on the race: on ontogenetic and on phylogenetic evolution.

I. *On the Individual.*—Disease may be defined as a “chemico-vital reaction to an inimical force which has broken through the first line of defence of the organism.” Alcohol readily breaks through the first line. As primary existing causes of chronic alcoholism, we have to consider not merely ethylic alcohol, but a number of other substances contained in alcoholic drinks, and then as consequences of the action of these, secondary toxic substances. Experimental observations have shown the evil effect of chronic alcoholism both on general immunity and on local resistance. The severity and variety of the lesions are largely due to bacterial toxæmias. The organism exhibits a general negative leucocyte chemio-taxis towards alcohol; and though it be granted that there occurs in the stomach, when alcohol is taken, a local leucocytosis (defensive in character), this is of short duration and insufficient to prevent the gradual development of gastric catarrh and atrophy in the inebriate. This atrophy leads to deficient secretion of mucus, which lowers defensive power, while the resulting enormous increase of saprophytic bacteria still further produces catarrh. There is a tendency to the production of myelocytic insufficiency in the bone marrow. Toxines are absorbed in greater amount than can be dealt with by the organism, and various systems become the subject of patho-

logical change. Delirium tremens is certainly Korsakow's psychosis, and peripheral neurites are probably dependent on secondary toxæmias.

The pathological changes are very varied, generally only a few of what may occur are present in a single case.

Among changes in the nervous system are mentioned:—*In the brain*: Wideness of sulci; thickening of pia-arachnoid; general pallor of cerebral substance; hæmorrhages and atrophic softening; sclerosis in first layer of cortex and in white matter; fibroid thickening, cellular increase, and hyaline changes in the vessels; apparent diminution in number of nerve cells; pigmentation of nerve cells; axonal type of degeneration in pyramidal cells (especially in Korsakow's psychosis); the presence of "ghost-cells" and intermediate varieties; secondary axis-cylinder degeneration. *In the cord* similar changes are found, besides large numbers of "amyloid bodies." In cases of *peripheral neuritis*: degeneration of fibres and thickening of arterioles and capillaries; reactive and degenerative change in nerve cells of posterior root ganglia and anterior horns of cord; degeneration of postero-internal columns in old-standing cases.

II. *On the Race*.—Largely through the investigation of Dr Beard, the processes connected with phylogenetic evolution have been brought into harmony with the cellular doctrine, and an antithetic alternation of generations has been established for the Metazoa. In view of all this, alcoholism can no longer be looked on as a cause of "racial evolution." The contentions of Dr Archdall Reid that diseases of parents do not affect the offspring through inheritance properly so called; "that temperance reform is impossible from the biological standpoint"; that "temperance reformers have failed because they have entered into a contest with nature"; and that "every scheme for the promotion of temperance which depends for success on the abolition or diminution of the alcoholic supply . . . is in effect a scheme for the promotion of drunkenness"—such contentions show a misunderstanding of the biological significance of disease.

Conjugation of dissimilar germs is not sufficient to explain genetic variations: environmental influences must be considered. Dr Reid maintains that there are reasons for doubting that germ cells can be injured by changes in parental blood and tissue produced by toxic substances, etc., and challenges proof that any influences—alcoholic or other acting on the parents—can cause variation in offspring. Now, recent researches on immunity have proved the remarkable responsiveness exhibited by somatic cells to external conditions, and that reaction changes in them may last for months or years. Such changes are of the nature of an adaptation to environment. One can see no valid reason for

doubting that germ cells can be similarly affected. In fact, Landsteiner, Metchnikoff, etc., have prepared specific spermotoxins, while Ceconi and Robecchi have succeeded in producing a specific ovarian cytotoxin. Biologically bacteria are germ cells, and we know how capable they are of being modified by environment. Among plants, also, various "sports" must be regarded as nutritional modifications of sporophytes, *i.e.* of germ cells. Professor Cossar Ewart's experiments with malarial pigeons afford evidence in the same direction. Selvatico-Estense records the case of a healthy woman married to a drunkard. She had five weakly children, all of whom died in infancy; while by a second husband of sober habits she had two healthy children.

Genetic variation results from the fact that germ cells are capable of being modified by environment as to potentialities of development. It is probable that the germ cells are most susceptible to such influences during the changes connected with reduction of chromosomes and ripening, at which time also the autonomy of the two nuclei (representing two ancestral lines) still present in the zygote and primary and secondary germ cells is lost. An essential object of gametic union is to check the tendency to variation and to fix the species. Tendencies to genetic variation produced by chronic alcoholism may be counteracted by influences from other ancestral lines of germ cells.

Dr Reid's position that if alcohol injuriously affected the germs, the effects would accumulate from generation to generation till the race would be extinct, cannot be maintained any more than his attempt to find in dipsomania an extreme "reversion to type." Evolution against disease is far from being simply an elimination of the inherently non-resistant. Infection or resistance are largely dependent on accidental circumstances; and "you cannot evolve against accident." Nor is it possible to eliminate those specially liable to become inebriates, because environmental causes of genetic variation (including alcohol) may in the next generation produce individuals of varying degrees of susceptibility in this respect.

Chronic alcoholism must be regarded as a most potent cause of genetic variation at the present day. The development of a "health conscience" becomes an all-important matter for the individual and the race.

A. HILL BUCHAN.

**THE HISTOLOGICAL APPEARANCES OF THE NERVOUS (155) SYSTEM IN EXPERIMENTAL HYDROPHOBIA.** CHARLES LADAME, *Journ. de Neurol.*, Feb. 20, and March 5, 1904.

THE certain diagnosis of hydrophobia as at present carried out has always been a lengthy and troublesome undertaking. And the

purpose of this paper is to determine if the histological appearances of the nervous system in animals dying of this disease are sufficiently characteristic to permit of a definite conclusion being arrived at by such an examination only.

A resumé is given of the more important observations published during the past thirty years regarding the pathological anatomy of rabies; and from this it appears that while some workers regard the changes recorded as specific, others maintain that similar appearances are to be met with in other diseases quite apart from rabies.

The author's personal observations include twenty-eight rabbits killed by a standard virus. In fourteen the injection was intracerebral, in nine subdural, and in five nasal. Each case is given in epitome; but the general conclusions arrived at may be briefly stated as follows:—The lesions of experimental hydrophobia as produced by a standard virus are *not* specific, and the cellular and vascular changes found throughout the nervous system are those common to any inflammatory process. The appearances vary greatly in individual animals, both as regards intensity and locality. Sometimes the lesions are confined to the cerebro-spinal ganglia, while again those ganglia may be little affected, and it is the cerebral membranes, or the cerebro-spinal axis, that show the principal changes. The mode of introduction of the virus does not appear to have any effect on the intensity or localisation of these lesions, and it is suggested that they depend rather on the resistance of the individual animals. Neither does the localisation nor the intensity of the lesions permit of a diagnosis of rabies being made, for similar appearances have been met with in other diseases. The cellular nodule found in the cerebro-spinal ganglia, and formerly held to be specific, is a form of inflammation peculiar to these ganglia, but it is met with in other conditions besides rabies. Chromatolytic changes in the pyramidal cells of cortex and spinal cord are rarely met with, and mostly where the hyperæmia and cellular exudation is most marked. Sometimes these cells are invaded by lymphocytes, which, however, seem, as far as one can see, to have but little effect on the general condition of the cell.

W. K. HUNTER.

**DESCRIPTION OF A PORENCEPHALIC BRAIN.** MARY BAIRD  
(156) HANNAY, *Glasgow Med. Journ.*, Vol. lxi. No. 3, March 1904.

THE brain is of small size, and is slightly wasted. There is marked asymmetry of the hemispheres, the left one being much smaller, and weighing 205 grammes less, than the right one. The corpus callosum and fornix are both very small, and the middle commis-



sure is represented by a mere tag of tissue. The general arrangement of sulci and gyri in the right hemisphere is natural. In the left hemisphere the frontal lobe is narrow, and a cavity extends across it, bounded mesially by the septum lucidum—which has a wide perforation—and having a lateral opening at about the usual site of Broca's convolution. The convolutions of the frontal lobe have a radial arrangement round the opening in front. Posteriorly the opening is bounded by the approximation of the central and temporal regions. The cavity has a lining of thickened ependyma, and is continued behind into the widely dilated left lateral ventricle. In its floor posteriorly, there is a prominence formed by the basal ganglia. The corpus callosum, fornix, and corpus albicans are all smaller than on the right side, and the foramen of Monro is wide. The basal ganglia are smaller, and are situated both farther back and on a lower plane than on the right side. The fissure of Sylvius cannot be traced with certainty beyond the posterior limit of the lateral opening of the cavity. The fissure of Rolando begins in the para-central lobule, and only extends for about half the distance between the great longitudinal and Sylvian fissures. The ascending limb of the intra-parietal sulcus is correspondingly short. On the lateral aspect of the frontal lobe there is only one distinct sagittal sulcus. The first temporal sulcus seems to end at the posterior limit of the lateral opening of the cavity. The central gyri are both exceedingly small and simple. They join one another below the short central fissure, a short transverse sulcus separating them from a rough irregular area which extends down to the lateral opening of the cavity. The first temporal and callosal gyri, and those of the superior parietal, quadrate, paracentral, and cuneate lobules, and of the posterior third or so of the first and second frontal gyri, are all very small. A small gyrus represents the insula. The cerebellum is comparatively of good size, and its hemispheres are of equal size and weight. The crus cerebri, pons and medulla are all smaller on the left than on the right side. On microscopical examination of sections from the left ascending frontal gyrus and para-central region, it is found that the cortex is very narrow (1.35 mm.), that the pyramidal cells are all very small, and that there is present a layer of granules. The cells in one of the radial gyri surrounding the lateral opening are of comparatively good size, but the cortex is as narrow as in the upper central region. In the first left temporal gyrus the cortex is of the same depth, but the cells are very small. In all of the above the neuroglia is natural. The rough area below the central fissure represents several gyri. The cortex measures only .75 mm. in depth, and the cells are extremely small. There is a superficial gliosis between contiguous gyri. In all the regions examined, there is perivascular and pericellular infiltration, and the larger pyramids show degene-

rative changes. From notes of the post-mortem, which was made several years ago, it is ascertained that the skull-cap was thickened and asymmetrical—smaller on the left side. The basal vessels were normal in size and arrangement, and there was no atheroma. The heart was normal. There was advanced phthisis pulmonalis, and the immediate cause of death was lobar pneumonia. The patient—a male—was a congenital imbecile. He was also epileptic. The right arm and leg were smaller than the left and were partially paralysed. (Paralysis was also congenital.) Death occurred at the age of thirty-seven years.

Six photographs illustrate the condition of the brain.

AUTHOR'S ABSTRACT.

### CLINICAL NEUROLOGY.

**POST-BASAL MENINGITIS DUE TO PNEUMOCOCCUS LANCEOLATUS (157) LATUS: RECOVERY.** PORTER PARKINSON, *Brit. Journ. of Children's Dis.*, Vol. i. No. 3, 1904, p. 112.

THE patient was a rachitic child aged two years, two months, who was admitted to hospital suffering from pneumonia and diarrhoea. The physical signs in the lungs persisted for about two months, ultimately resolving completely. About a fortnight after the beginning of the illness, cerebral symptoms, accompanied by a rise of temperature, set in. There was rigidity of the neck muscles, which went on to complete opisthotonos, with spastic flexion of the arms and extension of the legs. The opisthotonos remained for about five weeks, during which time the patient became blind. Eventually, however, all the symptoms subsided—in fact, the course of the illness was in every way characteristic of an ordinary case of post-basal meningitis. The interest of the case lies in the identification of the *diplococcus lanceolatus* instead of *d. intracellularis* as the causal organism in the cerebro-spinal fluid withdrawn by lumbar puncture, and in the fact that a meningitis, secondary to pneumonia, affected the base of the brain, instead of, as is usual, the vertex.

J. S. FOWLER

**SOME RARE PERIPHERAL PARALYSES.** BERNHARDT, *Berl. (158) klin. Wchnschr.*, March 7, 1904, p. 237.

BERNHARDT relates three cases of rare peripheral nerve lesions. The first was one of isolated paralysis of the supra-scapular nerve in a man of forty-one years of age. The patient complained of difficulty in lifting and carrying heavy loads with the affected arm.

Marked atrophy was found in the supra and infra spinati, while all the other muscles were healthy. A cervical rib was found on each side, but no definite relation could be made out between the presence of the abnormal rib and the nerve lesion. No definite cause could be found for the paralysis. In connection with this case, Bernhardt discusses the relations of cervical ribs to lesions of the brachial plexus.

Case II. was one of isolated paralysis of the musculo-cutaneous nerve following wounds made by a rope wound tightly round the upper arm. Paralysis of the biceps and brachialis anticus with corresponding disturbance of sensation was present. In addition there was marked weakness of the hand grasp, and some weakness in the triceps. In spite of these latter symptoms, Bernhardt regarded the case as one of injury limited to the musculo-cutaneous nerve.

Bernhardt also records an interesting case of paralysis of the left anterior crural and sciatic nerves, following the reduction of a congenital hip dislocation by Lorenz's method. The patient was a girl of eight, in whom the operation was followed by complete paralysis of the leg. The paralysis improved under treatment but did not completely recover. Bernhardt quotes Lorenz's views of the possibility of nerve injury in carrying out the manipulations for reducing a dislocation and refers to other recorded cases similar to his own.

He is of opinion that great care is necessary in reducing congenital dislocation of the hip, even in young children, to avoid nerve injury, and also that injury to the anterior crural nerve is less serious than injury to the sciatic nerve, as lesions of the anterior crural tend to recover more readily than lesions of the sciatic.

J. W. STRUTHERS.

**BERI-BERI IN MONKEYS.** HAMILTON WRIGHT, *Brain*, Winter (159) Number, 1903, p. 488.

THIS paper merits attention because of the light it throws upon a disease complex in its various types and of very uncertain etiology.

Beri-beri is due to an organism as yet undiscovered, which may be, or rather must be, ingested, but is not due to any special diet, and which is to be found in dark, damp places such as prison cells sometimes are. The organism is probably destroyed by sunlight. Having obtained entrance by the mouth, it develops in the stomach and duodenum, and there produces a toxin which passes into the blood stream and causes more or less extensive bilateral and symmetrical paralysis of certain afferent and efferent neurones.

Probably the active stage of development of the organism in the body is two to six weeks, and its incubation stage is seven to fourteen days.

There are three types of the disease :—

1. Acute pernicious beri-beri—in which the stomach and intestines show intense inflammation, the result of the organism, and a very potent toxin is produced in such an amount as to paralyse a large number of important afferent and efferent neurones, and so to cause death in one to seven days.

2. Acute and subacute beri-beri—in which a less acute and serious involvement of the stomach and intestines occurs, and a less amount of toxin is produced. These cases either, (a) after a partial paralysis of motor and sensory neurones, recover when the acute stage of the organism has ceased; or else (b) a partial sensori-motor paralysis follows, and remains for months or years, or the patient dies of cardiac exhaustion or from some intercurrent disease.

3. Beri-beric residual paralysis is really part of the preceding type, and is the stage of the disease when the effect only is present.

The author is doubtful as to whether the beri-beric poison acts centrally on the cells, peripherally on the neuronal terminations, or the neurones as a whole; but in the active stage of the disease he finds the cells and the terminal portions of the neuronal processes affected; while in the residual paralysis, if repair is not brought about, many degenerated nerve fibres are found in the main nerve trunks, although the distal ends of the posterior spinal ganglia and the ventral roots show no degeneration. The changes noted in certain of the cells of cord and medulla were very slight in cases of acute pernicious beri-beri, being mostly commencing chromatolysis, with later disappearance of Nissl's granules and a tendency towards an eccentric position of nuclei. In the residual paralysis of beri-beri there may be complete chromatolysis, the cell being either dense or clear, as if all the chromatic granules had disappeared; and in addition, the nucleus is more eccentric, and the nucleolus may have escaped from the cell. Together with this change in the cells, there is degeneration of the corresponding neuron processes. Lastly, in very long standing cases or where no repair is present, there may be vacuolation of the cell protoplasm, rupture of the cell and nucleolar membranes, with extrusion of the nucleolus and loss of some of the cell processes. Peripherally, very advanced and extensive degenerative changes occur in the neuron processes of the cells so affected.

The present series of experiments consisted in the confinement of four apparently healthy monkeys in the prison cells of prisoners who had died of the disease. A considerable time elapsed before any of them developed definite symptoms (one to four months).

The diet consisted of banana, pine-apple, and sugar-cane. The clinical features closely resembled those in man, although, unfortunately, one monkey became septic before it was killed.

A painstaking and careful microscopic examination was made of the medulla, cord, nerves, etc., in all cases, and special attention was paid to the cell groups affected in the different cord segments, and the degree of involvement of the vagus fibres which have a cardiac function. The stomach and duodenum were congested in all the four animals, and the affected nerve cells and neuronic processes corresponded to those affected in man.

The conclusions of this and preceding investigations by the author are the following:—

- (a) That beri-beri is an acute infectious disease.
- (b) That the organism as yet not known—exists in close, sunless places.
- (c) That no food as food is a factor in producing the disease.
- (d) That the organism is not one developing on or in the food of man.
- (e) That the organism is probably ingested accidentally with food.
- (f) That the organism multiplies in the stomach and upper part of the small intestine, causing local congestion, and that a toxin is elaborated which acts with varying power bilaterally and symmetrically on certain vital and ordinary neurones, giving rise to the symptoms known as beri-beri.
- (g) That the incubation period is short.
- (h) That rice is neither as diet nor as the habitat of a living specific organism, an etiological factor in producing the disease.

It only remains to add that several well drawn sketches of affected nerve cells conclude a very instructive paper.

ROBERT A. FLEMING.

**ALEXIA WITH ABILITY TO WRITE IN A CASE OF ACHON-**  
**(160) DROPLASIA AND IN AN IMBECILE.** RICHARD FOERSTER,  
*Rev. Neurol.*, 1903, p. 1206.

A MALE, 21 years of age, dwarfish, with enlargement of head and disproportionate development of limbs, had attended the school at Bicêtre for one year at the age of 18, after which time he was able to copy every text, written or printed, easily and without mistakes, but could not write without copy or to dictation, nor could he read what he had written. He was, however, familiar with a third of the letters of the alphabet, with three words, and the figures from 1 to 10; he was capable of drawing some simple familiar objects without model. Such cases of arrested develop-

ment are far from being rare; they are not, however, often met with in literature. A similar case was found in a quiet imbecile of 27 years. If early and carefully taught (before the fifteenth year, according to the experience of the observers at Bicêtre), such patients are certainly capable of much greater improvement.

HANS EVENSEN.

**THE MENTAL CONDITION IN EPILEPSY IN RELATION TO**

(161) **PROGNOSIS.** W. ALDREN TURNER, *Lancet*, April 9, 1904, p. 982.

1. A total of 161 cases, which have been under the writer's observation at the Colony for Epileptics, Chalfont-St-Peter, has been used for the investigation.

2. The mental features of the interparoxysmal state have been divided for descriptive purposes into four classes, the first containing those epileptics without any obvious mental impairment, and the fourth those with the highest grade of dementia.

3. The interparoxysmal mental condition is discussed in its several varieties and degrees with special reference to the influence of sex, hereditary disposition, age at onset, and character, duration and frequency of fits.

4. Sex has little influence upon the mental condition; but it may be stated in general terms that males are numerically more afflicted than females (91 per cent. of the former to 78 per cent. of the latter), but that the highest degree of dementia is somewhat more common in women than in men.

5. A family predisposition to epilepsy and insanity, although not necessarily militating against the retention of normal mental faculties, favours the supervention of some degree of mental impairment. A higher percentage with a hereditary history is found amongst those epileptics who show the profounder degrees of dementia than amongst those who show merely impairment of memory or a normal mental condition.

6. The duration of the disease influences to some extent the mental state. Mental impairment is less frequent when the fits have lasted for under five years, just as mental integrity is less commonly seen when attacks have persisted for ten or more years. But mental health and mental deficiency are observed in cases in which the disease has lasted, respectively, for over twenty years and for less than five years.

7. The age at the onset of the convulsions influences to some extent the subsequent mental condition, for the earlier the onset, especially during infancy and childhood, the less the probability of an unimpaired mental state.

8. The character and combination of the fits have an important relation to the mental condition, the profoundest degrees of dementia being most commonly seen when the major and minor attacks coexist. The petit mal seizures occurring alone are found with the lower grades of mental impairment. When the grand mal occurs alone, mental health is as common as mental deficiency.

9. There is a direct association between the frequency of the fits and the mental state, the more frequent the seizures the greater the degree of mental impairment and *vice versa*. Fits recurring in batches are accompanied by a high grade of dementia.

10. The term *facies epileptica* is applied to the physiognomy characteristic of the disease in many epileptics. It is found more commonly with the higher grades of dementia, but its existence is not limited to such cases, for it is observed in those with only impairment of memory as well as without any mental failure.

11. The view is upheld that the interparoxysmal mental condition seen in most cases of epilepsy is one expression of the neurosis of which the fits constitute another; that the former is therefore not directly dependent upon the seizures, and that the frequency and character—combination of the paroxysms in association with the degree of mental failure, indicate the severity and intensity of the disease.

12. The following figures show the percentage frequency of the four classes of mental deficiency amongst the 161 cases used for the purposes of this paper :—

Class A, intellectually normal.	.	.	13.6 per cent.
Class B, with impaired memory	.	.	31.6 "
Class C, feeble-minded	.	.	25.4 "
Class D, demented	.	.	29.1 "

#### AUTHOR'S ABSTRACT.

**IS EPILEPSY A FUNCTIONAL DISEASE?** M. ALLEN STARR,  
(162) *Journ. of Nerv. and Ment. Dis.*, March 1904, p. 145.

In this paper the author seeks to bring forward arguments to prove that epilepsy is usually, if not always, an organic disease.

His arguments are based on the study of 2000 cases of epilepsy and also on some other statistics regarding mal-development of the brain.

A. The author states it to be now generally accepted that Jacksonian epilepsy is due to organic changes in the brain. He regards the distinction between many cases of Jacksonian epilepsy and ordinary epilepsy to be one mainly of degree of severity and rapidity of extension of the cerebral irritation and consequent shock.

In his 2000 cases, 38 per cent. had auræ, while 23 per cent. had auræ of a distinctly Jacksonian type, so much so, that had general loss of consciousness not followed, these cases would have undoubtedly been placed in the category of Jacksonian epilepsy. This seems to indicate that these cases at least were due to an organic lesion.

*B.* The second argument is based on the study of 400 cases of mal-development of the brain; of these, 39 per cent. were subjects of epilepsy. In many cases of epilepsy the author considers that there are undoubted evidences of mal-development of the brain—evidences which may not be apparent to the parents of such cases, but which are obvious to the physician. In his opinion, it is the exception to find an epileptic in a perfectly normal mental and physical condition without signs of degeneracy or symptoms of some imperfect function. Thus, he considers that the signs of degeneracy merely indicate mal-development, and that the epilepsy is like these signs a manifestation of organic disease.

*C.* From a study of the supposed causes of epilepsy in his 2000 cases, he finds: (1) that a bad family inheritance—such as epilepsy, alcoholism in a parent, serious nervous disease, rheumatism, tuberculosis, etc., was present in 700 of his 2000 cases; (2) that trauma of the head was present in 229 cases out of his 2000; (3) that 50 cases came on after some acute infectious disease; (4) that the 36 cases of senile epilepsy were due to endarteritis and atheromatous changes in the vessels of the brain.

From among these supposed causes only those which have produced organic disease of the brain can be accepted as probable, and this he regards as another argument in favour of the disease being organic.

*D.* In the literature of epilepsy, many have found organic changes in cases of epilepsy, and he thinks that too little attention is paid to these observations.

He concludes that the attack of epilepsy is the symptom, not the disease; that its existence is proof of a weak and defective organisation of the brain; and that any lesion, no matter of what kind, is capable of interfering with the mechanisms of control which are present in the normal brain, and of giving rise to the symptom.

T. GRAINGER STEWART.

**THE OCCURRENCE OF HYPOTONIC FLAT FOOT IN GENERAL  
(163) PARALYSIS OF THE INSANE.** CH. FÉRÉ, *Nouv. Icon. de la Salpêtrière*, 1904, p. 79.

THE author points out the utility of the examination of imprints of the sole of the foot in clinical neurology. By this means one



can obtain a graphic record of the flattening of the foot, which results from fatigue and also of the progressive alterations which may take place in the plantar arch in nervous diseases. One can judge further of the rate of progress of certain nervous diseases by the change in the character of imprints of the feet taken at regular intervals.

The imprints are made by applying a thin coating of printers' ink to the sole of the foot, the patient subsequently placing the foot upon glazed paper in standing or walking, as occasion demands.

The chief points in the investigation may be summed up as follows :—

The peroneus longus is the muscle chiefly concerned in changes of the outline of the plantar arch.

Fatigue diminishes muscular tonicity, and the arch of the foot becomes markedly flattened. The same happens in neurasthenia, and in this malady the occasional occurrence of scoliosis and other deformities are considered to be the result of hypotonus.

Hypotonus is common in general paralytics. In one series of cases the author observed it in 28 per cent. and in another series in 16 per cent.

An excellent reproduction is figured of the imprints of the foot at an early and later stage of progressive general paralysis.

JAMES COLLIER.

**GRAPHIC STUDY OF THE PLANTAR REFLEX IN HEALTH (164) AND IN CERTAIN AFFECTIONS OF THE PYRAMIDAL SYSTEM.** HENRI VERGER and JEAN ABADIE, *Nouv. Icon. de la Salpêtrière*, 1904, p. 67.

THE authors consider that the plantar reflex can be resolved into three segmentary movements, simultaneous but distinct. The first of these occurs in the muscles moving the toes, and it is called the "planti-digital" reflex. The second occurs in the muscles of the leg which move the foot, and is called the "planti-tibial" reflex. The third occurs in the muscles which flex the leg upon the thigh, and which flex the thigh upon the pelvis, and to this the term planti-crural reflex. The planti-crural and planti-tibial reflexes were recorded by myographs placed directly upon the tensor fascia femoris and anterior tibial muscles respectively, while the planto-digital reflex was recorded by a tambour arrangement.

The following important facts are arrived at :—

1. As a result of a minimal stimulus applied to the plantar region, the planti-crural, planti-tibial and planti-digital reflexes appear invariably and simultaneously.

2. Two types of the planti-digital reflex are met with in normal subjects, (1) the type of pure flexion and (2) the type of flexion with consecutive slight extension.
3. The plantar reflex can only be elicited in the normal subject by stimulation of the plantar region, a condition strikingly different from that obtaining in certain pathological conditions.
4. Where lesions of the pyramidal tracts exist the planti-crural and planti-tibial reflexes differ from the normal only in their increased amplitude. The planti-digital reflexes on the other hand is met with in three types: (1) The type of pure flexion, comparable in every way with the normal; this type is sometimes met with in cases of severe spastic paralysis. (2) The type of flexion with consecutive extension; this differs markedly from the normal type of flexion with consecutive extension in that the flexion movement is of much shorter duration than the movement of extension, whilst in the normal type the reverse occurs. (3) The type of pure extension; the vivacity and amplitude of this type of reflex exceeds by far any normal type of the reflex. Any one of the pathological types of the planto-digital reflexes is not constantly associated with a special spastic affection of the pyramidal system.
5. The pathological plantar reflex is often obtainable not only by stimulation of the sole of the foot, but also by stimulation of the leg or even of any part of the lower limb.

JAMES COLLIER.

**A REFLEX ON THE DORSUM OF THE FOOT.** K. MENDEL,  
(165) *Neurol. Centralbl.*, March 1, 1904.

WITH a normal individual, when one percusses the side of the dorsum of the foot near the ankle, there follows dorsiflexion of all the toes save the big toe: the second or third toe usually shows the greatest dorsiflexion. The reflex is best obtained by supporting the inner edge of the foot and finding by percussion the spot on the outer half of the foot which gives the most lively reflex.

In functional nervous diseases the reflex is unchanged, and is all the clearer if the general reflex activity is exaggerated.

In some cases of organic nervous disease, instead of dorsiflexion, we have plantar flexion of the four small toes: in the majority of these cases Babinski's sign is present. In such cases there is a spastic paralysis of organic origin, and the reflex is the converse of Babinski's sign; but while it is almost always accompanied by

Babinski's sign, the latter is not always accompanied by Mendel's sign. The author looks for further investigation to determine the conditions of the appearance of his sign. While it is of great interest in connection with the whole question of reflexes, its diagnostic value is, as Mendel acknowledges, much reduced by the simultaneous presence of Babinski's sign.

C. MACFIE CAMPBELL.

## TREATMENT.

**THE FACIAL NERVE IN RELATION TO SURGERY.** BOCHEN-  
(166) HEIMER, *Arch. f. klin. Chirurg.*, Bd. 72, H. 3, 1904, p. 461.

BOCHENHEIMER details the results of a study of the topographical anatomy of the facial nerve from the stylo-mastoid foramen to its terminal distribution. His paper is divided into two sections. In the first he describes fully the course and relations of the seventh nerve beyond the stylo-mastoid foramen and the destination of each of its branches; and follows this by pointing out how the nerve and its twigs may best be avoided when making incisions in different parts of the face and neck.

In Section II. the author reviews the relations to the seventh nerve of the incisions made by surgeons in certain well-known typical operations in the facial area, such as those for exposing the divisions of the fifth nerve, for excising the upper jaw, etc., and he compares the advantages of certain alternative operations from the point of view of their avoidance or non-avoidance of the facial nerve branches.

The paper is illustrated by several figures illustrating the points emphasized in the text and gives a useful survey of the subject.

J. W. STRUTHERS.

---

# Bibliography

## ANATOMY

VAN GEHUCHTEN. Considérations sur la structure interne des cellules nerveuses et sur les connexions anatomiques des neurones. *Névraxe*, Vol. vi., f. 1, 1904, p. 81.

SOUKHANOFF. Contribution à l'étude du réseau endocellulaire dans les éléments nerveux des ganglions spinaux (par le procédé de Kopsch). *Névraxe*, Vol. vi., f. 1, 1904, p. 75.

- M. et Mme. DEJERINE. Le faisceau pyramidal direct. *Rev. Neurol.*, mars 30, 1904, p. 253.
- GEORGE DEAN and C. H. USHER. Experimental Research on the Course of the Optic Fibres. *Brain*, No. 104, Vol. xvi., p. 524.
- SAND. Beitrag zur Kenntnis der cortico-bulbären und cortico-pontinen Pyramidenfasern beim Menschen. *Arch. a. d. neurol. Instit. d. Wien. Univ.*, H. 10, S. 185.
- VAN GEHUCHTEN. Connexions centrales du noyau de Deiters et des masses grises voisines (Faisceau vestibulo-spinal, Faisceau longitudinal postérieur, Stries médullaires). *Névrose*, Vol. vi., f. 1, 1904, p. 19.
- MARBURG. Basale Opticuswurzel und Tractus peduncularis transversus. *Arch. a. d. neurol. Instit. d. Wien. Univ.*, H. 10, S. 66.
- HATSCHEK. Zur Kenntnis der Pedunculus corporis mammillaris, des Ganglion tegmenti profundum und der dorso-ventralen Raphesfaserung in der Haube. *Arch. a. d. neurol. Instit. d. Wien. Univ.*, H. 10, S. 81.

#### COMPARATIVE ANATOMY—

- REDLICH. Zur vergleichenden Anatomie der Associationsysteme des Gehirns der Säugetiere. I. Das Cingulum. *Arch. a. d. neurol. Instit. d. Wien. Univ.*, H. 10, S. 104.
- ZUCKERKANDL. Zur vergleichenden Anatomie des Hinterhauptlappens. *Arch. a. d. neurol. Instit. d. Wien. Univ.*, H. 10, S. 297.
- GRÜNWALD. Zur vergleichenden Anatomie der Kleinhirnarms. *Arch. a. d. neurol. Instit. d. Wien. Univ.*, H. 10, S. 368.
- DAVID HEPBURN and DAVID WATERSTON. A Comparative Study of the Grey and White Matter of the Motor-Cell Groups, and of the Spinal Accessory Nerve, in the Spinal Cord of the Porpoise. *Journ. Anat. and Physiol.*, April 1904, p. 295.
- JOHN CAMERON. On the Presence and Significance of the Superior Commissure throughout the Vertebrata. *Journ. Anat. and Physiol.*, April 1904, p. 275.
- HATSCHEK. Über eine eigentümliche Pyramidenvariation in der Säugetierreihe. *Arch. a. d. neurol. Instit. d. Wien. Univ.*, H. 10, S. 48.
- JOHN CAMERON. On the Origin of the Epiphysis Cerebri as a Bilateral Structure in the Chick. *Proc. Roy. Soc. Edin.*, Vol. xxv., No. 2, 1904, p. 160.

#### PHYSIOLOGY

- HITZIG. Physiologische und klinische Untersuchungen über das Gehirn. Hirschwald, Berlin, 1904.
- MATIEGKA. Über die Bedeutung des Hirngewichts beim Menschen. *Anat. Hefte*, Bd. 23, H. 3, 1904, S. 655.
- CARLO PARIANI. Sul contegno delle cellule nervose del simpatico, dei gangli plessiformi e dei nuclei centrali del vago nella pneumonite sperimentale. *Riv. di Patolog. nerv. e ment.*, Marzo 1904, p. 120.
- MOSSO. Action des centres spinaux sur la tonicité des muscles respirateurs. *Arch. ital. de Biol.*, Vol. xli., 1904, p. 138.
- CHARPENTIER. Généralisation, par les voies nerveuses, de l'action des rayons N appliqués sur un point de l'organisme. *Compt. rend. Acad. d. sc.*, No. 11, 1904, p. 715.
- DENIS COURTADE. Rapport entre la contraction volontaire et les réactions électriques neuro-musculaires. *Bull. de la Soc. Française d'Electrother.*, mars 1904, p. 92.
- SHINKISHI HATAI. A Note on the Significance of the Form and Contents of the Nucleus in the Spinal Ganglion Cells of the Fetal Rat. *Journ. Comp. Neurol. and Psychol.*, March 1904, p. 27.
- JOHN GORDON WILSON. The Relation of the Motor Endings on the Muscle of the Frog to Neighbouring Structures. *Journ. Comp. Neurol. and Psychol.*, March 1904, p. 1.
- DEBRAY. Quelques déductions pratiques de la réfutation du neurone. *Journ. de Neurol.*, mars 20, 1904, p. 101.
- E. B. MEIGS. On the Mechanism of the Contraction of Voluntary Muscle of the Frog. *Am. Journ. Med. Sc.*, April 1904, p. 667.
- DE BEULE. Le mécanisme des mouvements respiratoires de la glotte chez le lapin. *Névrose*, Vol. vi., f. 1, 1904, p. 1.
- E. S. LONDON. Action des rayons du radium sur le sens de la vue. *Arch. d'Electricité méd.* avril 10, 1904, p. 243.

ALICE JOHNSON and EDWIN GOODALL. On the Action of the Blood Serum from Cases of Acute Mental Disorder on *B. Coli Communis*. *Brit. Med. Journ.*, April 9, 1904, p. 826.

### PSYCHOLOGY

- SPITZKA. A Study of the Brain of the late Major J. W. Powell. *Am. Anthropol.*, Vol. v., No. 4, p. 585.  
 SCHOLZ. Die moralische Anästhesie. E. H. Mayer, Leipzig, 1904, M. 3.60.  
 VASCHIDE et PELLETIER. Recherches expérimentales sur les signes physiques de l'intelligence. Montligeon, 1904.  
 GRASSET. Pensée et Cerveau. La doctrine biologique du double psychisme et le spiritualisme. Montligeon, 1904.  
 GOLDSCHIEDER. Ueber das Vibrationsgefühl. *Berl. klin. Wchnschr.*, April 1904, S. 853.  
 E. G. SPAULDING. An Establishment of Association in Hermit Crabs, *Eupagurus longicarpus*. *Journ. Comp. Neurol. and Psychol.*, March 1904, p. 49.  
 ROBERT M. YERKES. Space Perception of Tortoises. *Journ. Comp. Neurol. and Psychol.*, March 1904, p. 17.  
 PIERRE BONNIER. Le sens des Attitudes. Naud, Paris, 1904.

### PATHOLOGY

- BERNHARDT. Neuropathologische Betrachtungen und Beobachtungen. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 26, H. 1-2, 1904, S. 78.  
 HANKE. Das Gehirn eines congenitalen, bilateralen Anophthalmus. *Arch. a. d. neurol. Instit. d. Wien. Univ.*, H. 10, S. 58.  
 FUCHS. Die Veränderungen der Dura mater cerebialis in Fällen von endokranieller Drucksteigerung (Tumor und Hydrocephalus), nebst einem Beitrag zur Histologie der Dura mater spinalis. *Arch. a. d. neurol. Instit. d. Wien. Univ.*, H. 10, S. 378.  
 WAGNER. Ueber Verimpfung von Karzinom in das Gehirn von Versuchstieren. *Wien. klin. Wchnschr.*, März 24, 1904, S. 323.  
 OBERSTEINER. Über das hellgelbe Pigment in den Nervenzellen und das Vorkommen weiterer fettähnlicher Körper im Centralnervensystem. *Arch. a. d. neurol. Instit. d. Wien. Univ.*, H. 10, S. 245.  
 JAMES COLLIER and E. FARQUHAR BUZZARD. The Degenerations Resulting from Lesions of Posterior Nerve Roots and from Transverse Lesions of the Spinal Cord in Man. *Brain*, No. 104, Vol. xxvi., p. 559.  
 GIUNIO CATOLA. Contributo allo studio del fascio piramidale nell'emiplegia cerebrale infantile. *Riv. di Patolog. nerv. e ment.*, Marzo 1904, p. 113.  
 ORLOFF. Zur Frage der pathologischen Anatomie der genuinen Epilepsie. *Arch. f. Psychiat.*, Bd. 38, H. 2, 1904, S. 439.  
 W. FORD ROBERTSON. The Pathology of Chronic Alcoholism. *Brit. Journ. Inebriety*, April 1904, p. 226.

### CLINICAL NEUROLOGY AND PSYCHIATRY

#### GENERAL—

- MAURICE DE FLEURY. Manuel pour l'étude des maladies du système nerveux. Félix Alcan, Paris, 1904, 25 fr.  
 HERSMAN. The Influence of Typhoid Fever on the Nervous System. *N. Y. Med. Journ.*, March 26, 1904, p. 598.

#### MUSCLES—

- JAMIN. Experimentelle Untersuchungen zur Lehre von der Atrophie gelähmter Muskeln. Fischer, Jena, 1904, M. 5.  
 WOLFSTEIN. A Case of Atrophy of the Hand Muscles, with Localised Sensory Disturbances; possible Early Syringomyelia. *Journ. Am. Med. Assoc.*, April 9, 1904, p. 941.  
 Muscular Dystrophy.—P. PINI. Distrofia muscolare congenita a forma ipertrofica. *Ric. crit. di Clin. Med.*, Aprile 9, 1904, p. 237.

#### PERIPHERAL NERVES—

- MAX LEMKE. Beitrag zum Regenerationsvorgang im peripheren Nerven. *Arch. f. Psychiat.*, Bd. 38, H. 2, 1904, S. 490.

- RAYMOND. Les localisations du cancer sur le système nerveux périphérique. *Arch. de Neurol.*, avril 1904, p. 273.
- BUCKLEY. Brachial Neuritis. *Lancet*, April 16, 1904, p. 1042.
- GERMAN. Über Neuritis mit besond. Berücksicht. der Neuritis migrans, nach Beobachtungen aus der medicin. Poliklinik zu Göttingen. Vandenhoeck & Ruprecht, Göttingen, 1904, M. —80.
- PASCAL. Contribution à l'étude de la polynevríte alcoolique; Diagnostic; Psychose polynevrétique. Firmin, Montane et Sicardi, Montpellier, 1904.
- MERKLEN et GUIARD. Paralysie saturnine à type partiel Aran-Duchesne. *Arch. de Neurol.*, avril 1904, p. 309.
- Beri-beri.—HAMILTON WRIGHT. Beri-beri in Monkeys. *Braia*, No. 104, Vol. xxvi, p. 488.

#### SPINAL CORD—

- BIER und DÖNITZ. Rückenmarksanästhesie. *München. med. Wchnschr.*, April 5, 1904, S. 593.
- Friedreich's Ataxia.—BING. Die Abnützung des Rückenmarks (Friedreichsche Krankheit und Verwandtes). *Deutsche Ztschr. f. Nervenheilk.*, Bd. 26, H. 1-2, 1904, S. 163.
- Tabes.—FLATAU. Die Tabes dorsalis. Konegen, Leipzig, 1904, M. 1.40.
- A. C. BRUCH. The Etiology of Tabes Dorsalis. *Journ. Am. Med. Assoc.*, April 2, 1904, p. 870.
- KIERNAN. Forensic Eye Aspects of Tabes. *Med. News*, March 19, 1904, p. 555.
- STEMBO. Beseitigung sehr heftiger lancinirender Schmerzen bei einem Tabiker nach 28 Injectionen von antirabischer Markemulsion. *Neurol. Centralbl.*, April 1, 1904, S. 303.
- BERNADICON. Contribution à l'étude des rapports symptomatiques entre le tabes et l'hystérie. *Thèse de Paris*, 1904.
- KOLLARITS. Zur Kenntnis der tabetischen Arthropathie und Knochenerkrankung. *Neurol. Centralbl.*, April 1, 1904, S. 304.
- Multiple Sclerosis.—TOTSCH. Weitere Beiträge zur Kenntnis der multiple Sclerose des Hirns und Rückenmarkes. *Prag. med. Wchnschr.*, März 24, 1904, S. 147.
- ROSENFELD. Endarteritis bei multipler Sklerose. *Arch. f. Psychiat.*, Bd. 38, H. 2, 1904, S. 474.
- BYROM BRAMWELL. Clinical Lecture on Disseminated Sclerosis with Special Reference to the Frequency and Etiology of the Disease. *Clin. Studies*, April 1904, p. 193.
- MASSALONGO. Sclerosi a placche familiari. Contributo alla patogenesi delle paraplegie spasmodiche eredo-famigliari. *Riv. crit. di Clin. Med.*, Aprile 9, 1904, p. 233.
- Pseudo-Sclerosis.—FRANKL-HOCHWART. Zur Kenntnis der Pseudosklerose. *Arch. a. d. neurol. Instit. d. Wien. Univ.*, H. 10, S. 1.
- Tumours of Cauda Equina.—G. C. DAVIS. Tumours involving the Cauda Equina; with Report of Case. *Journ. Am. Med. Assoc.*, March 19, 1904, p. 751.
- Myelitis.—SAXL. Über einen Fall von Kompression-myelitis der Wirbelsäule bei Wirbelcaries. *Arch. a. d. neurol. Instit. d. Wien. Univ.*, H. 10, S. 230.
- Spine.—COMPTON RIELY. A Study of the Anatomy, Pathology and Etiology of Scoliosis, also presenting a Scoliotone, an Apparatus for Elongating and Lessening the Rotation of the Spine in Lateral Curvature. *Journ. Am. Med. Assoc.*, April 2, 1904, p. 872.
- KILBURN. Eye-strain considered as a Factor in Production of Lateral Curvature of the Spine. *Boston Med. and Sur. Journ.*, March 24, 1904, p. 318.
- LOVETT. Forcible Correction in Lateral Curvature of the Spine. *Boston Med. and Sur. Journ.*, March 17, 1904, p. 283.

#### BRAIN AND MENBRANES—

- JOHNSON SYMINGTON. John Gratton's Craniometer and Craniometric Methods. *Journ. Anat. and Physiol.*, April 1904, p. 259.
- HÖLSCHER. Die otogenen Erkrankungen der Hirnhäute. Marhold, Halle, 1904, M. 1.
- WILLIAM N. BULLARD. Astereognosis in Cerebral Affections. *Journ. Nerv. and Ment. Dis.*, April 1904, p. 241.
- W. ROSSELL JUDD. A Case of Lead Encephalopathy. *Brit. Med. Journ.*, April 16, 1904, p. 888.
- Meningitis.—G. W. SQUIRES. A New Sign of Basilar Meningitis. *Med. Rec.*, March 26, 1904, p. 496.

- MARFAN, AVIRAGNET et DETOT. Méningite hémorragique subaiguë avec hydrocéphalie chez les nouveau-nés. *Bull. méd.*, No. 5, 1904.
- RENAUD. Accidents et complications des méningites cérébro-spinales. *Thèse de Paris*, 1904.
- KARL V. WIEG. Zur Klinik der Meningitis tuberculosa. *Neurol. Centralbl.*, April 16, 1904, S. 343.
- Hæmorrhage.**—J. HOGARTH PRINGLE. An Unusually Situated Hæmorrhage from the Meningeal Artery. *Scot. Med. and Sur. Journ.*, April 1904, p. 328.
- Hemiplegia.**—CAMPBELL THOMSON. Associated Movements in Hemiplegia: their Origin and Physiological Significance. *Brain*, No. 104, Vol. xxvi., p. 514.
- KINNIER WILSON. The State of the Eye Muscles in Organic Hemiplegia. *Rev. Neurol. and Psychiat.*, April 1904, p. 285.
- Diplegia.**—NATHAN. A Case of Cerebral Diplegia (so-called "Spastic Spinal Paralysis"). *N. Y. Med. Journ.*, March 26, 1904, p. 581.
- Progressive Muscular Atrophy.**—F. W. MOTT. Two Clinical Lectures on Progressive Muscular Atrophy. *Lecture I. Practitioner*, April 1904, p. 488.
- H. M. FLETCHER and F. E. BATTEN. Case of Progressive Muscular Atrophy of Spinal Origin in a Girl aged 11 years; with Autopsy. *Brain*, No. 104, Vol. xxvi., p. 488.
- Bulbar Paralysis.**—WILFRED HARRIS. Post-Diphtheritic Chronic Bulbar Paralysis, and its distinction from Myasthenia. *Brain*, No. 104, Vol. xxvi., p. 548.
- Cerebral Softening.**—HENNEBERG. Ueber chronische progressive Encephalomalacie und Bemerkungen über den harten Gaumenreflex. *Arch. f. Psychiat.*, Bd. 38, H. 2, 1904, S. 609.
- Cerebral Tumours.**—W. G. SPILLER. Report of Five Cases of Tumor of the Brain. *Am. Journ. Med. Sc.*, Feb. 1904.
- W. J. TAYLOR. Report of a Case of Tumor of the Brain, an Endothelioma of the Dura; Operation, Recovery, Death in 108 days from Recurrence of the Disease. *Am. Journ. Med. Sc.*, Feb. 1904.
- RINDFLEISCH. Über diffuse Sarkomatose der weichen Hirn- und Rückenmarkshäute mit charakteristischen Veränderungen der Cerebrospinalflüssigkeit. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 26, H. 1-2, 1904, S. 185.
- CHAROËT et BANCEL. Gliome volumineux au lobe frontal gauche. *Lyon Méd.*, avril 10, 1904, p. 717.
- Empyema of Frontal Sinus.**—PAUL S. MERTINS. Empyema of Frontal Sinus, followed by Extrasural Abscess and Abscess of Frontal Lobe; Operation and Death from Hypostatic Congestion of the Lungs. *Am. Journ. Med. Sc.*, April 1904, p. 684.
- Cerebral Syphilis.**—NEUMANN. Beitrag zur Kenntnis der Hirusyphilis. *Wien. med. Wchnschr.*, April 9, 1904, S. 622.
- LUTHER PETER. The Diagnosis and Treatment of Syphilis of the Central Nervous System. *Med. News*, March 12, 1904, p. 494.
- GEORGE OGILVIE. What is Syphilitic Disease of the Nervous System? *Clin. Journ.*, April 3, 1904, p. 401.
- General Paralysis.**—SÁVAGE. Two Lectures on General Paralysis of the Insane. *Clin. Journ.*, March 30, 1904, p. 369.
- CORNILLOT. De la perte de mémoire du calcul comme signe précoce de l'affaiblissement intellectuel chez les paralytiques généraux. *Thèse de Paris*, mars 1904.
- MÖBIUS. Progressive Paralyse und berühmte Leute. *Psych.-neurol. Wchnschr.*, Nr. 1, 1904.
- Cortical Lesions.**—CAMPBELL THOMSON. A Case of Injury to the Cerebral Cortex (Clinical Study). *Clin. Journ.*, March 30, 1904, p. 392.
- BONHOEFFER. Über das Verhalten der Sensibilität bei Hirnrindenläsionen. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 26, H. 1-2, 1904, S. 57.
- Fracture of Skull.**—LOUIS BATHE RAWLING. Hunterian Lectures on Fractures of the Skull. *Lect. I. Lancet*, April 9, 1904, p. 973.
- O. WATERMAN and B. POLLACK. Fracture of the Basis Cranii. *Journ. Nerv. and Ment. Dis.*, April 1904, p. 250.

#### GENERAL AND FUNCTIONAL DISEASES—

- MONIN. Les Névropathes. R. de Rudeval, Paris, 1904, 3 fr. 50.
- Chorea.**—JAMES BURNET. The Association of Chorea with Tonia Solium. *Brit. Journ. Children's Dis.*, April 1904, p. 147.
- Paralysis Agitans.**—RUHEMANN. Ueber Schüttellähmung nach Unfällen. *Berl. klin. Wchnschr.*, März 28, April 4 u. 11, 1904.
- Epilepsy.**—W. B. SPRATLING. Psychologic Aspects of Epilepsy. *Medicine*, Feb. 1904.

- PICK. Ueber eine besondere insidïöse durch das Fehlen der Krampfanfälle charakterisierte Form des Status epilepticus. *Wien. klin. Wchschr.*, März 24, 1904, S. 331.
- W. ALDREN TURNER. The Mental Condition in Epilepsy in Relation to Prognosis. *Lancet*, April 9, 1904, p. 982.
- CHAS. J. ALDRICH. Epileptic Automatism. With Report of a Case. *Am. Med. Compend.*, March 1904.
- I. H. NEFF. Report of a Case of Myoclonus Epilepsy. *Am. Journ. Insan.*, Jan. 1904.
- WILLIAM P. SPRATLING. Guides to the Prognosis in Epilepsy, with Remarks on the Curability of the Disease; including Reports of 34 Cases. *N.Y. Med. Journ.*, April 9, 1904, p. 681.
- WILDERMUTH. Über die Aufgaben des Pflegepersonals bei Epileptischen. Marhold, Halle, 1904.
- Hysteria.**—BRATZ. Hysterie und Epilepsie. *Arch. f. Psychiat.*, Bd. 38, H. 2, 1904, S. 500.
- Neurasthenia.**—RICHARDSON. Neurasthenia. *Med. News*, March 12, 1904, p. 547.
- Neuralgia.**—SICARD. Névralgie du trijumeau et ponction lombaire. *Soc. de biol.*, mars 4, 1904, p. 357.
- Exophthalmic Goitre.**—HENRY POSERT. Exophthalmic Goitre. Résumé of Cases and their Treatment. *Memphis Med. Monthly*, March 1904.
- ARTHUR W. ROGERS. Graves' Disease and its Relation to the Psychoses. *Medicine*, March 1904.
- Cretenism.**—WEYGANDT. Der heutige Stand der Lehre vom Kretinismus. Marhold, Halle, 1904.
- WEYGANDT. Ueber Virchow's Cretenentheorie. *Neurol. Centralbl.*, April 1 u. 16, 1904, S. 290.
- BAYON. On So-called Intra-uterine Cretenism and its Supposed Identity with Endemic and Sporadic Cretenism. *Lancet*, April 16, 1904, p. 1051.
- BAYON. Ueber das Centralnervensystem der Cretenen. *Neurol. Centralbl.*, April 16, 1904, S. 388.
- SELIGMANN. Cretenism in Calves. *Journ. of Pathol.*, March 1904, p. 311.
- Infantilism.**—BYROM BRAMWELL. Case of Pancreatic Infantilism: Results of Treatment. *Scot. Med. and Sur. Journ.*, April 1904, p. 321.
- J. F. CLARKE. Two Cases of Infantile Myxœdema. *Journ. Am. Med. Assoc.*, March 19, 1904, p. 768.
- Erythromelalgia.**—ARTHUR S. HAMILTON. A Case of Erythromelalgia. *Journ. Nerv. and Ment. Dis.*, April 1904, p. 217.

#### IN TAL DISEASES—

- SIEMERLING. Psychiatrie im Wandel der Zeiten. Lipsius & Tischer, Kiel, 1904, M.—60.
- H. CAMPBELL THOMSON. Cerebral and Mental Diseases in Relation to General Medicine: A Review of Recent Literature. *Practitioner*, April 1904, p. 561.
- GEORGE STOCKTON. The Relation of Physical Defects to Mental Development. *Med. Rec.*, March 1904, p. 412.
- JOHN MACPHERSON. Variation in its Relation to the Origin of Insanity and the Allied Neuroses, being the Morrison Lectures for 1904. *Rev. Neurol. and Psychiat.*, April 1904, p. 235.
- KRONTHAL. Nervenzellen und Psychose. *Arch. f. Psychiat.*, Bd. 38, H. 2, 1904, S. 625.
- BERKHAN. Über den angeborenen und früh erworbenen Schwachsinn. Vieweg & Sohn, Braunschweig, 1904, M. 2.40.
- KLIX. Über Geistesstörungen in der Schwangerschaft und im Wochenbett. Marhold, Halle, 1904, M. 1.
- J. R. KNAPP. Notes on Malignant Growths in the Insane. *Am. Journ. Insan.*, Jan. 1904.
- HAMMERSCHLAG. Über Hörstörungen im Kindesalter und ihre Bedeutung für die psychische und intellektuelle Entwicklung des Kindes. *Wien. med. Presse*, April 3 u. 10, 1904, S. 658.
- STADELMANN. Das Wesen der Psychose. Heft 1. Das psychische Geschehen. Ballhorn & Cramer, Würzburg, 1904.
- HALBERLAH. Über die acute Commotions-psychose, zugleich ein Beitrag zur Aetiologie des Korsakow'schen Symptomencomplexes. *Arch. f. Psychiat.*, Bd. 38, H. 2, 1904, S. 402.



- H. W. MILLER. Korsakoff's Psychosis. Report of Cases. *Am. Journ. Insan.*, Jan. 1904.
- PENTA. In tema di demenza arterio-sclerotica. *Riv. mens. di psichiat. forense*, Anno vii., Num. 2-3, 1904, p. 83.
- C. W. PAGE. Paranoid Dementia. *Am. Journ. Insan.*, Jan. 1904.
- M'DONALD. The Present Status of Paranoia. *Amer. Journ. Insan.*, Jan. 1904.
- RICHARD DEWEY. Apparent Recovery in a Case of Paranoia. *Am. Journ. Insan.*, Jan. 1904.
- MONZARDI. Contributo allo studio delle inversioni sessuali. *Riv. mens. di psichiat. forense*, Anno vii., Num. 2-3, 1904, p. 49.
- P. BLUM. Étude d'un cas de rypophobie. *Arch. gén. de méd.*, mars 29, 1904, p. 778.
- LUGARO. L'ipocondria persecutoria. Una forma tardiva della demenza paranoide. *Riv. di Patolog. nerv. e ment.*, Marzo 1904, p. 105.
- BEDUSCHI. La Catistofobia (acatisia di Haskovec) come sindrome psicastenica. *Riv. di Patolog. nerv. e ment.*, Marzo 1904, p. 124.
- Criminal Psychiatry.**—MAZEN. Contribution à l'étude de la simulation de la folie chez les criminels. Thèse, Firmin, Montane et Sicardi, Montpellier, 1904.
- PENTA. La follia nelle carceri. *Riv. mens. di psichiat. forense*, Anno vii., Num. 2-3, 1904, p. 67.
- Forensic.**—HOCHÉ. Zur Frage der Zeugnisfähigkeit geistig abnormer Personen. Marhold, Halle, 1904.
- Treatment.**—FRANCIS WARNER. Training of the Feeble-Minded. *Lancet*, March 26, 1904.
- WEYGANDT. Die Verhütung der Geisteskrankheiten. Stuber, Würzburg, 1904, M.—75.
- DIECKHOFF. Die Anwendung der Isolierung bei der Behandlung Geisteskranker. *München. med. Wchnschr.*, März 29, 1904, S. 550.
- JEAN CAMUS et PH. PAGNEZ. Isolement et Psychothérapie. Traitement de l'hystérie et de la neurasthénie. Félix Alcan, Paris, 1904, 9 fr.
- ALT und VORSTER. Gutachten über die Bezirks-Irrenanstalt zu Saargemünd. *Arch. f. Psychiat.*, Bd. 38, H. 2, 1904, S. 693.

**ALCOHOL, ETC.—**

- S. B. LYON. Some Drug and Alcoholic Habitués. *N. Y. Med. Journ.*, April 2, 1904, p. 644.
- PFÄFF. Die Alkoholfrage vom ärztlichen Standpunkt. F. Pietzcker, Tübingen, 1904, M.—80.
- KASSOWITZ. Der Arzt und der Alkohol. M. Perles, Wien, 1904, M. 1.20.
- G. H. R. DABBS. A Note on the Inebriate in Making. *Brit. Journ. Inebriety*, April 1904, p. 278.
- SHEARMAN. The Mental Development of the Inebriate. *Brit. Journ. Inebriety*, April 1904, p. 257.
- HARRY CAMPBELL. Pre-Alcoholic Peoples. *Brit. Journ. Inebriety*, April 1904, p. 221.
- CROTHERS. The Study of Inebriety in America. *Brit. Journ. Inebriety*, April 1904, p. 281.
- NORMAN MEACHEN. The Cutaneous Affections of the Inebriate. *Brit. Journ. Inebriety*, April 1904, p. 274.
- MEYER. Ueber acute und chronische Alkoholpsychosen und über die ätiologische Bedeutung des chronischen Alkoholmissbrauches bei der Entstehung geistiger Störungen überhaupt. *Arch. f. Psychiat.*, Bd. 38, H. 2, 1904, S. 331.
- ROBERT JONES. The Relation of Inebriety to Mental Disease. *Med. Press*, April 13, 1904, p. 388.
- LLOYD TUCKEY. The Position of Hypnotic Treatment in the Cure of Chronic Alcoholism. *Brit. Journ. Inebriety*, April 1904, p. 268.
- PLAVEC. Die Therapie der akuten Phosphorvergiftung. *Wien. med. Presse*, März 20, 1904, S. 563.
- KÖSTER. Ein klinischer Beitrag zur Lehre von der chronischen Schwefelkohlenstoffvergiftung. *Deutsche Zechr. f. Nervenheilk.*, Bd. 26, H. 1-2, 1904, S. 1.

**CRANIAL NERVES—**

- J. HERBERT PARSONS. The Arris and Gale Lectures on the Neurology of Vision. *Lancet*, April 16, 1904, p. 1029.
- H. WRIGHT THOMSON. Three Cases of Ocular Paralysis. *Glasgow Med. Journ.*, April 1904, p. 276.
- HATSCHEK. Sehnervenatrophie bei einem Delphin. *Arch. a. d. neurol. Instit. d. Wien. Univ.*, H. 10, S. 223.

- HENRI DUFOUR. Déviation conjuguée de la tête et des yeux avec hémianopsie homonyme; son origine sensorielle. *Rev. Neurol.*, avril 15, 1904, p. 833.
- J. R. NELSON. Asthenopia and Headache from Eye-strain. *Med. Rec.*, March 19, 1904, p. 453.
- KOLLARITS. Über Migraine ophthalmoplégique. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 26, H. 1-2, 1904, S. 128.
- STEFANI. Comment se modifie la réaction de la pupille à l'atropine à la suite de l'usage prolongé de cette substance. *Arch. ital. de Biol.*, Vol. xli, 1904, p. 1.
- TSCHIRJEV. Ein Fall vollständig geheilter Blindheit (Hemianopsie). *Arch. f. Psychiat.*, Bd. 38, H. 2, 1904, S. 459.
- KREUZFUCHS. Über den Dilatationsreflex der Pupille auf Verdunklung. *Arch. a. d. neurol. Inst. d. Wien. Univ.*, H. 10, S. 275.
- LANDAU. Drei Fälle von halbseitiger Atrophie der Zunge (Hemiatrophia linguae). *Deutsche Ztschr. f. Nervenheilk.*, Bd. 26, H. 1-2, 1904, S. 102.

## REFLEXES—

- KUTTNER. Die nasalen Reflexneurosen und die normalen Nasenreflexe. Hirschwald, Berlin, 1904, M. 6.

## MISCELLANEOUS SYMPTOMS—

- SANGER BROWN. A Neurologic Clinic; Spasmodic Torticollis, etc. *Med. Standard*, March 1904.
- CHEINISSE. La paralysie périodique familiale. *Semaine méd.*, avril 13, 1904, p. 113.
- STUART McDONALD. A Case of General Streptothrix Infection. *Scot. Med. and Surg. Journ.*, April 1904.
- WILLIAM BRUCE. Referred Pains, mostly Gouty in Origin. *Scot. Med. and Surg. Journ.*, April 1904, p. 297.
- MONRO and MCGREGOR. A Chronic Neurosis characterised by Frequent Paroxysms of Pain, Swelling, and Hæmorrhage in Various Parts of the Body; Death from Acute Pulmonary Tuberculosis. *Lancet*, April 16, 1904, p. 1089.
- GWILYM DAVIS. Ludwig's Angina. Report of a Case. *Am. Journ. Med. Sc.*, Feb. 1904.
- CHAS. J. ALDRICH. Leuconychia Striata Arsenicalis Transversus; with report of Three Cases. *Am. Journ. Med. Sc.*, April 1904, p. 702.

## TREATMENT—

- BÜDINGEN. Eine neue Methode zur Prüfung therapeutischer Massnahmen bei Rückenmarkskrankheiten. *München. med. Wchnschr.*, März 29, 1904, S. 558.
- NEUMANN VON SCHÖNFELD. Stärkere Nerven. Schule des Willens und die Wach-Autosuggestion nach der Nancyer Methode. Nermann, Zürich, 1904, M. 4.
- GASTON LYON. Pathogénie et traitement des névroses intestinales. Masson et Cie, Paris, 1904, 1 fr. 25.
- YVERT. Traitement du tic douloureux de la face. *Gaz. Méd. de Paris*, mars 19 et 26, 1904, p. 145.
- Surgical.**—LÉNORMANT. Chirurgie de la tête et du cou. Masson et Cie, Paris, 1904, 4 fr. 50.
- NIMIER. Blessures du crâne et de l'encéphale par coup de feu; chirurgie du système nerveux. Félix Alcan, Paris, 1904, 15 fr.
- ANDREW B. ROSS. Treatment of a Case of Depressed Fracture of the Skull caused during Birth. *Brit. Med. Journ.*, April 16, 1904, p. 880.
- HUTCHINSON. Intracranial Resection of the Second Division of the Fifth Nerve for Epileptiform Neuralgia. *Lancet*, April 16, 1904, p. 1044.
- Electrical.**—MANN. Elektrodiagnostik und Elektrotherapie. Hölde, Wien, 1904.
- CLUZET. Sur l'excitation des nerfs par décharges de condensateur. *Journ. Physiol. et Pathol. gén.*, T. 6, No. 2, 1904, P. 204.
- LAQUERRIÈRE et MOREL. Contribution à l'étude de l'influence des traitements électriques sur les échanges chez les Basedowiens. *Bull. de la Soc. Française d'Electrother.*, mars 1904, p. 87.
- PERTHES. Die Bedeutung der Röntgenstrahlen für die Diagnose und Operation der Stirnhöhlenosteome. *Arch. f. klin. Chir.*, Bd. 72, H. 4, 1904, S. 1022.
- DUBOIS. Notes sur le Traitement électrique du Goitre exophtalmique. *Bull. d'Electrother.* No. 1, 1904, p. 22.
- H. THIELLE. Neurasthénies traitées par les courants continus et le franklinisation. *Bull. d'Electrother.*, fév. 1904, p. 44.

# Review of Neurology and Psychiatry

---

## Original Articles

### **HEREDITARY SYPHILITIC TABES (JUVENILE TABES).**

By R. T. WILLIAMSON, M.D. (Lond.), F.R.C.P.,  
Physician, Ancoats Hospital, and Assistant Physician, Royal Infirmary,  
Manchester.

**TABES dorsalis** is exceedingly rare under the age of 20, but a number of cases are on record in medical literature, in which the disease commenced in childhood or youth. Such cases have been described as infantile or juvenile tabes, or hereditary syphilitic tabes; and though they are extremely rare, the number on record is steadily increasing. The reports of the cases published show that usually the parents have suffered from syphilis, or the tabetic children have presented signs or symptoms of hereditary syphilis; in other cases of juvenile tabes the patient has been infected with syphilis at an early period of life, often through a syphilitic nurse (extra-genital syphilitic infection). In some cases one of the parents of the young tabetic patient has suffered both from syphilis and tabes. The following records are abstracts of notes on three cases of juvenile tabes, which have been under my care at the Ancoats Hospital and Manchester Royal Infirmary.

CASE I. F. C., girl aged 8 years (was sent to me by Dr J. G. Clegg of the Manchester Royal Eye Hospital in February 1903). The sight had been failing for five months (possibly much longer). There had been no headache, no vomiting, and no

other symptoms, except shooting pains in the limbs, which were noticed first about the time when the sight began to fail. On the first examination (in February 1903) with the ophthalmoscope, primary double optic atrophy was detected, along with patches of disseminated choroiditis in left fundus. The vision was markedly impaired, especially in the left eye, which had been affected first. The pupils did not react to light. There were frequent irregular movements of the eye-balls (nystagmus of blindness). The knee-jerks and tendo Achilles reflexes were both absent, and the patient complained of stabbing pains in the legs.

During the last twelve months I have frequently examined the case. The patient is now (March 1904) quite blind. The optic discs present the typical appearances of primary optic atrophy. There are well-marked patches of disseminated choroiditis in the left fundus. The pupils are dilated and equal; they do not react either to light or accommodation. There is no paralysis of the ocular, facial or tongue muscles, and no paralysis of the limbs. The irregular movements of the eye-balls (nystagmus of blindness) are now very slight. The knee-jerks and tendo Achilles reflexes are absent. The wrist-jerks and triceps-jerks are absent. The patient continues to suffer from stabbing pains in the feet and legs, which present the characteristic features of the pains of tabes. [The pains are severe and stabbing, they are of short duration, and are frequently repeated. The pains are separated by painless periods. Some days the pains are severe and frequent; at other times they cause little discomfort, and for several or many days may be very slight.] Ataxia, hypotonus, anæsthesia, and other signs of tabes are all absent.

The absent patella and Achilles reflexes, the shooting pains, the absence of pupillary reflexes, and the primary optic atrophy clearly indicated early tabes. The disseminated choroiditis in the left fundus suggested congenital syphilis.

The family history was important. I was informed that the father was suffering from spinal disease. I visited him, and found that he presented the symptoms and signs of advanced tabes dorsalis.

*History of patient's father* (now 45 years of age).—In 1882 he suffered from syphilis, which was followed by definite specific

secondary symptoms. About 1895 he commenced to suffer from shooting pains in the legs (which were characteristic of those of tabes). About 1900 the sight began to fail. Optic atrophy was diagnosed. (The patient had been a watchmaker and there had been great and prolonged eye strain in his work.) About 1901 he commenced to suffer from gastric crises. In 1902 he became quite blind.

When I examined him (March 1904), there was marked ataxia of the arms and legs, but no paralysis of the limbs. He was quite unable to stand, owing to ataxia. The knee-jerks were absent. He complained of a girdle sensation, of frequent shooting pains in the limbs, and of attacks of vomiting (gastric crises). He was completely blind. Ophthalmoscopic examination revealed marked primary double optic atrophy. The pupils were unequal,  $R < L$ ; they did not react to light.

The child's *mother* appeared healthy and presented no signs of tabes, but she had had four miscarriages. She had then been carefully treated medically, and the fifth child was the patient whose history has been just recorded. The sixth child is a boy aged 7 years, who appears healthy. His knee-jerks and tendo Achilles reflexes are present. The pupils react to light and there are no shooting pains and no signs of tabes.

CASE II. John H., aged 13 years, was sent to me by Dr Taylor of Bolton. Early in 1903, failure of sight had been noticed. For a few months previous to the visual failure there had been a little unsteadiness in walking. There had been no headache, no vomiting, no discharge from the ears. For three years he had suffered from "aching" pains in the feet.

When he first came under treatment in Bolton in September 1903, Dr Taylor had detected early optic atrophy: vision was then  $\frac{5}{16}$  in. in the right eye,  $\frac{5}{16}$  in the left. On December 11, 1903, vision in the right eye was reduced to  $\frac{5}{16}$  (Dr Taylor). When I first examined the patient in December 1903, the knee-jerks and tendo Achilles reflexes were absent. The pupils did not react to light. The boy suffered frequently from pain in the feet. Ophthalmoscopic examination revealed double primary optic atrophy. The boy's upper central incisor teeth were both notched and presented the typical appearances of the teeth of congenital syphilis (Hutchinson's teeth). The boy's father had lived a

reckless life and had been separated from his wife for many years. The patient's mother had had eleven children. The first four were born dead; one child lived five days only; one child lived seven months; five children are still alive.

In March 1904, I again examined the boy. There was slight ataxia. He was unable to stand alone on one leg with eyes closed. The pupils did not react to light. The knee-jerks and tendo Achilles reflexes were absent, and the patient suffered from pains in the feet. The optic discs presented the appearances of primary optic atrophy. The patient was quite blind.

On April 23rd, the patient was unsteady when standing with his feet together and eyes closed. He was unable to stand on one leg. In walking he was a little unsteady, but this might be partially attributed to his visual failure. Slight inco-ordination could be detected in the arm movements when he attempted to touch the tip of the nose, or to bring the tips of the index fingers together with the eyes closed. The knee-jerks and tendo Achilles reflex were absent. The plantar reflexes were present and of the normal type. Ankle clonus was absent. There was muscular hypotonus in the legs; he was able to raise the legs, with the knees extended, up to an angle of  $90^\circ$ , when placed in the horizontal position. There was no anaesthesia. The left pupil was a little larger than the right. There was no reaction of the pupils to light. Nystagmoid movements of the eyeballs (in a lateral direction) occurred frequently (nystagmus of blindness).

CASE III. Minnie M., girl aged 17 years, came under treatment on account of loss of sight and pains in the legs. Failure of sight had commenced at the age of 10 years. Since the age of 13 she had been almost blind. There had been no headache, no vomiting, and no signs of brain tumour.

On *examination*, in April 1904, no objects held in front of the eyes could be recognised, but there was a very slight perception of light. Ophthalmoscopic examination revealed marked primary double optic atrophy. The right pupil reacted very slightly to a powerful electric light; the left pupil did not react. Both pupils reacted to accommodation.

There was no paralysis of ocular, facial, or tongue muscles.

There was marked myopia in each eye ( - 24 D in the R., - 28 D in the L.).

There was no paralysis of limbs. The patient was able to walk alone, but there was great ataxia. Rhomberg's symptom was well marked. The knee-jerks, tendo Achilles reflexes, and the wrist and triceps jerks were all absent. There was no plantar reflex.

No anæsthesia of the legs and no bands of diminished sensation on the chest could be detected.

There was a slight lateral curvature of the spine (convexity to the left) and slight pes cavus.

The patient complained of frequent severe, sharp, shooting pains in the legs, which exactly corresponded in their character with the shooting pains of tabes.

The teeth did not present any indications of congenital syphilis. The girl's father admitted that he had suffered from syphilis. The mother had had several miscarriages.

The father (in April 1904) did not suffer from ataxia or pains in the legs; his knee-jerks were present; the tendo Achilles reflex was present on the right side, absent on the left. The pupils were unequal  $L > R$ , but both reacted to light. Apart from the absence of the right tendo Achilles reflex and the inequality of pupils, the father did not present any signs of tabes.

Though the spinal curvature and the slight pes cavus are symptoms which occur in Friedreich's disease, this affection could be definitely excluded on account of the optic atrophy and the shooting pains in the legs, which are not symptoms of the affection just mentioned. Also a plantar reflex of the extensor type (Babinski's reflex) was not obtained in the case here recorded; in Friedreich's disease this type of plantar reflex is common.

\* \* \* \* \*

Cases of juvenile tabes have been recorded and referred to by Sir Wm. Gowers, Strümpell, Remak, Homén, Oppenheim, and others.

Halban (Abstract, *Neurologisches Centralblatt*, No. 1, 1903) records a case of juvenile tabes in a patient aged 23. As a child the patient was infected with syphilis through his nurse.

Maas (Abstract, *Review of Neurology and Psychiatry*, April 1903) records five cases of tabes in early life. The patients were from 16 to 22 years of age when the first symptoms

appeared. In three of the cases there were definite signs of congenital syphilis.

Idelsohn (Abstract, *Neurologisches Centralblatt*, No. 1, 1903) reports a case of infantile tabes in a girl of 6. The parents were syphilitic.

Rad records two cases of tabes in young persons—one in a girl aged 10, the other in a boy aged 7. In the latter case there was evidence of congenital syphilis; in the former, hereditary syphilis was very probable.

Babinski (*Soc. Med. des Hôpitaux*, Oct. 24th, 1902, see *Journ. of Nervous and Mental Diseases*, p. 186, 1903) records two cases of hereditary syphilitic tabes—one that of a young woman aged 22, the other that of a girl aged 15. Both patients presented signs of hereditary syphilis, and the father of both suffered from tabes. Babinski states that about 20 cases of hereditary tabes are on record. In the discussion on these cases, Sonques referred to a family of four persons (parents and two daughters): the father died of general paralysis, the mother suffered from tabes, both the daughters presented symptoms of tabes.

Hartmann (*Münchener med. Woch.*, No. 51, 1903) records the case of a female aged 20, who had suffered since the age of 13 from neuralgic pains in the face, and since the age of 17 from impaired vision, due to optic atrophy. There was also Rhomberg's symptom and loss of knee-jerks. The patient was an illegitimate child, and had probably suffered from hereditary syphilis.

Linser (*Münchener med. Woch.*, No. 15, 1903) has collected 21 cases of juvenile tabes recorded in medical literature. In 17 of these there was a clear history of syphilis in the parents; in two this history was probable, and in two doubtful. Optic atrophy was a common symptom, but ataxia was absent in two-thirds of the cases.

Erb has also recently referred to most of the cases on record (*Berliner klin. Woch.*, Nos. 1-4, 1904).

Briefly stated, the points of interest in the three cases which I have recorded are as follows:—

First Case, girl aged 8; signs of early tabes; father had



suffered from syphilis, and now presents the symptoms of advanced tabes; mother has had four miscarriages.

Second Case, boy aged 13; signs of early tabes; teeth characteristic of congenital syphilis (Hutchinson's teeth).

Third Case, girl aged 17; symptoms and signs of tabes for 7 years; father has had syphilis; mother has had several miscarriages.

All three patients were blind, owing to primary optic atrophy. In two cases there was evidence of congenital syphilis; in the third case, congenital syphilis was probable, the father having suffered from syphilis.

---

### **A CASE OF SENILE COMBINED SCLEROSIS OF THE SPINAL CORD.<sup>1</sup>**

By S. A. KINNIER WILSON, M.A., M.B., B.Sc., and  
Dr OCTAVE CROUZON (of Paris).

THE term "combined sclerosis" of the spinal cord is used to designate, not a clinical entity, but the anatomico-pathological grouping of certain morbid conditions characterised by the presence of sclerotic changes both in the posterior and in the lateral columns. It is accordingly the pathological substratum common to several clinical types.

These may be classified as follows:—

1. Congenital and family combined scleroses.
  - (a) Friedreich's disease.
  - (b) Hereditary cerebellar ataxia (Pierre Marie).
  - (c) Spastic paraplegia occurring in families (Strümpell).
2. Acquired combined scleroses.
  - (a) Combined scleroses of the tabetic type.
  - (b) Combined scleroses occurring in general paralysis.
  - (c) Spastic combined sclerosis.
  - (d) Combined scleroses in old age (associated with arterio-scleroses).
  - (e) Subacute combined scleroses (in anæmias, intoxications and cachexias) (1).

<sup>1</sup> A brief resumé of this case was presented to the Neurological Society of Paris, March 3rd, 1904.

The literature, more especially on the subject of senile combined scleroses, is still scanty; it is true that the diagnosis, from the clinical point of view, has been made in a certain number of cases, but the anatomical verification of the condition has frequently been awaiting. The case which we venture to place on record may accordingly not be without interest.

The patient was a man 68 years of age, who became an inmate of the Hospice of Bicêtre—under the care of Professor Pierre Marie—only a day or two before his death, and the clinical observation of his condition was unfortunately, though necessarily, incomplete. His general state was very low: he had sores on his heels and on his sacral region; he suffered from incontinence of urine and of feces. For a long time he had been able to walk only with the help of crutches, but the nature of this trouble of gait—whether it was acquired or due to the malformation of his left tibia, which was markedly curved—was not elicited. He presented at the same time a funnel-shaped depression of his thorax at the level of the xiphisternum.

At the autopsy, the spinal cord was found to be remarkably small; the anterior and posterior roots were likewise small and slender. The posterior meninges were distinctly thickened, especially in the upper part of the dorsal region.

The cord was hardened in a mixture of formalin and Müller's fluid. After three weeks it was transferred to Müller alone. Celloidin sections of all the segments were prepared in the usual way, and stained by the methods of Weigert, of Weigert-Pal, and of Weigert-Pal counterstained with cochineal.

This histological examination revealed the following condition:—

*Lumbar region.*—The posterior columns are intact. In the lateral columns there is a slight sclerosis in the areas occupied by the pyramidal tracts, extending to the periphery of the cord. There is no alteration in the zones of Lissauer.

*Inferior dorsal region.*—At this level a very slight sclerosis is discernible in the posterior columns, stretching in an antero-posterior direction from the posterior commissure to the border of the section. The sclerosed area is in shape not unlike an hour-glass, whose two bases correspond to the anterior and to the posterior parts of the column, while its "waist" corresponds to the middle of the posterior median septum.

In the lateral columns, the sclerosis in the pyramidal tracts is both more marked and more distinctly limited, the direct cerebellar tracts being untouched. At the same time the area of degeneration has a tendency to spread in a forward direction, following the curve of the margin of the cord.

*Middle dorsal region.*—The sclerosis in the posterior columns is more pronounced, though the affected area has changed its shape. Anteriorly, the sclerosis is intense; it is limited to a narrow band bordering the posterior median septum in its ventral part. Posteriorly, the sclerosis is slighter and more diffuse, its lateral limits being formed by two symmetrical fibrous septa which pass in from the periphery of the cord. The narrow band of intense sclerosis just described appears as it were to penetrate sharply into this latter more diffuse area, well towards the posterior border of the cord.

As far as the lateral columns are concerned, there is no great change from the preceding levels: the sclerosis continues to spread forward parallel to the margins.

*Upper dorsal region.*—In the dorsal part of the posterior columns the sclerosis remains diffuse, though fairly intense. Bordered laterally by fibrous septa, as described above, it follows their direction only for a short distance, continuing forwards independently in a remarkably straight line; the sclerosis may therefore be said to be systematised.

Ventrally, the sclerosis is more and more limited to the median septum. It may be described as having the appearance of a lance head, the base corresponding to the posterior commissure, and the fine drawn out point piercing the posterior diffuse sclerotic area.

It is to be remarked that this lance-head area is definitely more sclerotic than the other affected parts of the columns of Goll.

The examination of serial sections from below upwards shows that the point of this area approaches the posterior margin of the cord inferiorly; as one passes up, it retreats proportionately further and further from the periphery.

At this level there is an evident change in the lateral column appearances.

*Inferior cervical region.*—Here the sclerosis of the posterior columns, more and more systematised, is practically limited to

the posterior part of the columns of Goll, while the anterior lance-head zone similarly undergoes a modification. It begins to retreat from before backwards from the posterior commissure, while its point is much less distinctly marked off from the surrounding sclerosis; in fact it tends to become assimilated with the diffuse sclerotic areas in the posterior part of the tracts of Goll.

Laterally, the sclerosis touches the margin of the cord on both sides, invading the area occupied by the direct cerebellar tracts; it continues to advance in a crescent shape anteriorly.

*Superior cervical region.*—The lance-head zone is lost in the diffuse sclerosis of Goll's columns. The lateral condition is as before. In the anterior columns—on one side only—there is a very slight sclerosis in the area occupied by the direct pyramidal tract; it is limited to the border of the anterior median fissure, and does not spread out in the crescent shape described by Marie and Guillain.

*Medulla, pons, and cerebrum.*—The degeneration in the columns of Goll extends into the lower part of the medulla. The sclerosis in the crossed pyramidal tracts has now become less distinctly marked, while that in the direct pyramidal tract is still in evidence. But as one passes gradually upward, all this pyramidal sclerosis diminishes as gradually in intensity, and careful examination of sections of the cerebral peduncles fails to reveal any lesion or pathological change in the pyramidal tracts.

As far as the cerebrum is concerned, no cortical or sub-cortical lesion was found.

One or two further details of the microscopical examination remain to be considered.

As far as one may gather from the sections stained with cochineal, there was no gross alteration in the cells of the anterior horns. We failed similarly to find any definite evidence of posterior or anterior root lesions.

Last of all, sections from every segment of the cord were systematically examined for vascular changes. No appreciable alteration or modification from the normal sufficient to amount to a pathological condition could be detected.

To sum up this detailed description, we find a combined sclerosis of the posterior and lateral columns of the cord. The sclerosis in the latter extends from the lumbar levels to the



FIG. 1.

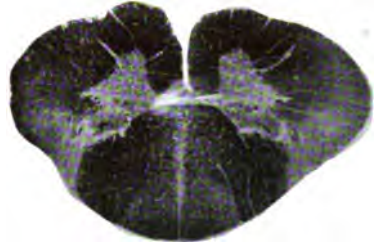


FIG. 2.

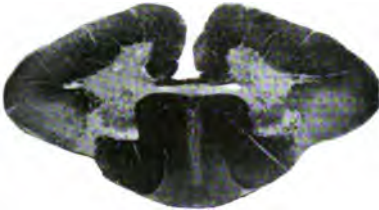


FIG. 3.



FIG. 4.

#### DESCRIPTION OF FIGURES.

(After sections coloured by the method of Weigert-Pal.)

FIG. 1.—*Upper lumbar region*.—There is distinct sclerosis in the areas occupied by the two crossed pyramidal tracts. Notice also the fainter degeneration—not unlike an hour-glass—in the posterior columns.

FIG. 2.—*Inferior dorsal region*.—The pyramidal sclerosis is more marked. The posterior portions of the columns of Goll are degenerated, while a more sharply defined wedge of sclerosis penetrates into them from before backwards.

FIG. 3.—*Upper dorsal region*.—The pyramidal degeneration is extending forwards following the margins of the cord, though the direct cerebellar tracts remain distinct. The zone *en fer de lance* has retreated from the periphery of the cord, remaining sharply differentiated from the more diffusely sclerosed area in the posterior part of the columns of Goll.

FIG. 4.—*Inferior cervical region*.—The degeneration in the lateral columns extends well forwards in a crescentic shape, though the fibres of the direct cerebellar tracts still bound it postero-laterally. In the posterior columns the sclerosis is diffuse and limited to the columns of Goll, while the more definite sclerotic zone is now reduced to a very small oval elongated area on either side of the posterior median septum about its middle.



medulla, with its maximum in the upper dorsal region. In the former, beginning in the inferior dorsal segments, it increases as one passes up, and can be followed also to the medulla. In the areas under consideration (lateral and posterior) the sclerosis is apparently systematised: on the one hand occupying the pyramidal zones throughout, scarcely reaching the periphery except over a small section of the cord; on the other, being confined to the columns of Goll, and to the ventral zone of the peculiar shape already described.

A consideration of this case from the clinical and from the anatomico-pathological standpoints seems to render legitimate its classification in the category of conditions, still comparatively rare, known as senile combined scleroses of the spinal cord. We are indebted for our knowledge on this subject to the work of Demange (2), Reverchon (3), Pic and Bonnamour (4), Hirsch (5), and Crouzon (6).

In 1884, under the name of "progressive tabetic contracture," Demange described a condition which he had observed in old subjects with evidence of arterial degeneration. This condition supervenes late in life (in his cases it appeared gradually in women who had already been in hospital some time on account of advancing old age). Walking becomes more difficult, the legs become stiff and heavy, gradually definite contracture sets in. The tendon reflexes, at first exaggerated, eventually diminish. Sensibility remains intact, and there is no loss of sphincter control. The evolution of the disease is subacute, death usually supervening after some months. This clinical description is based by Demange on four cases, in each of which subsequent pathological examination revealed an anatomical substratum corresponding to the clinical picture. The lesions were confined exclusively to the spinal cord: in no case was there any evidence whatever of cerebral involvement. In his own words, "the lesion consisted in a diffuse disseminated sclerosis of the cord, vascular in origin, limited more specially to the white matter, but possibly involving the grey, simulating at the same time a systematised combined sclerosis."

Atheroma of the spinal vessels is the underlying morbid process according to Demange, yet the paralytic affections one sees in atheromatous old men cannot always be attributed to spinal conditions, and cerebral vascular change may give rise to

symptoms which must be distinguished from those of "progressive tabetic contracture."

More recently Pic and Bonnamour, and Reverchon in his thesis (under the direction of Professor Pic), have given analogous descriptions under the name of "spastic paresis in atheromatous old men." Their clinical type is characterised by a spastic gait, accompanied by contracture, increased knee-jerks, epileptoid tremor, and sometimes an extensor response. Pathologically one finds evident spinal lesions confined to the lateral and posterior columns.

According to Pic and Bonnamour, the sclerosis is attributable to a pial thickening; it appears to begin at the periphery of the cord, and perhaps vascular alterations have something to do with the condition. The sclerosis is not really systematised; it only appears to be so. They consider the conclusion justifiable that this "spastic paresis" is the result of a medullary (*i.e.* spinal) and not a cerebral lesion.

Hirsch has about the same time published an article on arterio-sclerosis of the spinal cord. The symptomatology of this condition is, according to him, motor rather than sensory, a reason for this clinical differentiation being found in the fact that the vascular supply of the anterior part of the cord is less complete than that of the posterior. Nevertheless, he admits that posterior column lesions have sometimes been predominant; thus in his series of eight cases, five clinically bear a resemblance to tabes.

Finally, Crouzon, from his clinical examination of various cases of the type of disease under consideration occurring in old men housed in the Hospice of Bicêtre, has made a tentative classification of the condition into "pareto-spastic," "ataxo-spastic," and "ataxo-cerebello-spastic" groups. In spite of the slight distinctions which serve to differentiate these types clinically, it is none the less possible to recognise in them certain characters in common. Thus the condition begins in later life or old age (the age of four of his cases, 50, 52, 60 and 69). Its evolution is slow and progressive (duration of condition in these four cases, 2, 6, 9 and 15 years respectively). Syphilis is denied in all; it may almost certainly be left out of account. In two of his cases Crouzon found troubles of speech.

The motor difficulties were confined to the lower limbs; in



one of the patients, however, there was an intentional tremor of the upper extremities, not unlike what is seen in disseminated sclerosis.

From this brief account of our present knowledge on the question of senile combined sclerosis, it will be seen that here clinical observation in a sense outweighs as yet anatomical and pathological fact. This appears to us sufficient justification for the recording of our case, though from the clinical point of view it is admittedly incomplete, and we cannot with any definiteness ascribe to it its proper place in the classification given above. It certainly differs very distinctly from the combined sclerosis of the tabetic type which we have observed, which are for the most part associated with meningeal and lymphatic affections in the cord, and which are further pseudo-systematised. The lesions in our case are certainly systematised, however, both for the lateral and for the posterior columns.

The statement has already been made that in our case no distinct evidence is forthcoming of vascular degeneration, and in this it differs notably from preceding descriptions (*cf.* more especially those of Demange, Pic and Bonnamour) where the lesions appeared consecutive to atheroma of the spinal vessels. In spite of this, however, the absence of any cerebral condition, coupled with the age of the patient, would suggest the diagnosis we make. It is certain that the underlying morbid condition of senile combined sclerosis is not yet thoroughly elucidated; and it is at least conceivable that a pathological process such as must have been at work in our case need not necessarily betray itself by vascular alteration.

In any case, not merely from the anatomico-pathological, but also from the clinical point of view, senile combined sclerosis cannot be said to be as definitely recognised as some of the other forms, *e.g.* the tabetic; and it is to be hoped that later investigation will not merely add to the facts, but aid in their systematisation and classification.

There is one point to which we should like to draw attention in the consideration of our case, *viz.*, the singular sclerotic area in the posterior columns, more especially in the dorsal region, which in our note to the Neurological Society of Paris we described as resembling a slender lance head (*zone en fer de lance*) penetrating by its drawn-out extremity the more diffusely

sclerosed area in the columns of Goll. Beginning in the inferior dorsal region, and clinging from its commencement to the posterior median septum, more especially in its ventral part, it gradually diminishes on ascending, first from behind forwards, and then from before backwards, till in the inferior cervical region it is reduced to a mere point and disappears. This zone does not correspond exactly to any intramedullary root path, as far as we know; besides, though the posterior roots were found at the autopsy to be slender, later examination failed to make evident any degeneration. It does not correspond either to any of the recognised systematised bundles of the posterior columns, its dorsal position in the cord being remembered.

Quite recently Nageotte (7), in an anatomical study of the posterior columns, has figured this same zone in a case of lesion of the roots of the cauda equina without ascribing to it much significance (see his Obs. I.). According to his description, there exists (in his case) in the middle and upper dorsal regions, in the anterior half of the column of Goll, a sort of notch (*encoche*) in the otherwise diffusely sclerosed column, by which a number of non-degenerated fibres penetrate into this sclerosed column in a backward and inward direction, thus appearing to limit the median degenerated zone corresponding to the one figured by us. For him, the area which corresponds to our zone *en fer de lance* is merely a division of the sclerosed column of Goll, separated from the posterior part by some sound fibres. As a matter of fact, the careful examination of our case shows that the zone in question is more distinctly and intensely sclerosed than the rest of the posterior columns: it is not simply limited as described by Nageotte, for we do not find any "invasion of healthy fibres"; on the contrary, the rest of the columns of Goll is very diffusely and equally sclerosed.

In our opinion, the area is most probably systematised. Its exact significance is at present doubtful: its possible relation to or connection with endogenous fibres—a question by no means as completely elucidated for the dorsal and cervical as for the dorso-lumbar, lumbar and lumbo-sacral divisions—is a matter for future investigation.

Our thanks are due to Professor Pierre Marie for his kind permission to record the case.

## BIBLIOGRAPHY.

1. O. Crouzon. "Des Scléroses combinées de la moelle," *Thèse de Paris*, 1904.
  2. Demange. *Rev. de Méd.*, 1884-5.
  3. Reverchon. *Thèse de Lyon*, 1902.
  4. Pic and Bonnamour. *Rev. de Méd.*, Jan. and Feb. 1904.
  5. Hirsch. *Journ. Nerv. Ment. Dis.*, Feb. 1903.
  6. Crouzon. *Loc. cit.*
  7. Nageotte. *Nouv. Icon. de la Salpêtrière*, Jan.- Feb. 1904.
- 

## Abstracts

## ANATOMY

**THE DIRECT PYRAMIDAL TRACT.** M. and Mme. DEJERINE, (167) *Rev. Neurolog.*, March 1904, p. 253.

THIS article is written in criticism of a paper by Marie and Guillain in *La Semaine Médicale* (1903, No. 3), in which they endeavoured to show that the direct pyramidal tract consisted of two sets of fibres, one derived from the cortex and one from the mesencephalon. According to these authors, the amount of degeneration of the direct pyramidal tract caused by cortical lesions is insignificant, and occupies but a small sectional area of the spinal cord in the postero-internal part of the anterior median column; it is further said to be limited to the cervico-dorsal region of the cord. That part of the tract which is supposed to arise in the mesencephalon, on the other hand, is said to be a voluminous bundle extending the whole length of the cord, and occupying a crescentic area anterior to the cortical portion. If these statements are true, it is possible from an examination of a degenerated direct pyramidal tract to say whether the degeneration is due to a lesion of the mesencephalon or of the mantle.

The authors of the present paper consider that these conclusions are not warranted by the facts. They reproduce numerous sections of cord from patients who suffered from lesions of the cortex and corona radiata, which sections show degeneration of the greater part of the sectional area of the direct pyramidal tract. They further reproduce sections of cord from patients who had suffered from lesions of the mesencephalon, sections which show extensive degeneration of the bulbar pyramid, but very little degeneration of the direct pyramidal tract.

While the authors admit that the fibres of the pyramidal tract above the decussation are mixed up with pallio-tectal fibres and

with ascending fibres from the various basal ganglia, and that the fibres of the direct pyramidal tract in the cord are mixed up with fibres derived from the reticular formation, the posterior longitudinal bundle and the nucleus of Deiters and with endogenous fibres of the cord, they deny that any fibres derived from the basal ganglia pass through to the direct pyramidal tract.

M. and Mme. Dejerine suggest that Marie and Guillain have arrived at erroneous conclusions by failing to recognise the normal variability in the amount of decussation of the pyramidal tracts which takes place in the bulb.

W. H. B. STODDART.

**CONCERNING A PECULIAR PYRAMIDAL VARIATION IN**  
(168) **MAMMALS.** OBERSTEINER, *Arch. a. d. Neurol. Inst. a d. Wiener Univ.*, H. x., 1903, p. 48.

OBERSTEINER in recent papers has described some important variations in the course of the pyramidal fibres in man. He made the suggestion that these variations may be accounted for by the lateness of development of the pyramidal fibres, which must therefore occupy any position which is left for them.

Hatschek undertook the examination of the nervous system of other mammals to ascertain the existence and frequency of such variations in them. These were found, but in the brain of *pteropus edulis* H. found a remarkable and hitherto undescribed condition of which he gives a detailed account in his paper.

Starting from the upper end of the cord, sections were examined through the medulla, pons, etc.

Two abnormalities are fully described and discussed:—

1. At the level where the accessory olive appears, the pyramidal fibres have lost their peripheral and ventro-lateral position, and have become crushed out dorso-laterally, the accessory olive being now ventro-lateral and coming right up to the surface. This dorso-lateral position of the pyramidal fibres at the level of the olive has been found and described in other animals, *e.g.* cetaceans. In them, however, an explanation is offered in the large development of the accessory olive, which pushes out the pyramidal fibres laterally and comes to the surface. This will not altogether account for the condition in *pteropus*.

He is inclined to believe that the condition in *pteropus* may be the normal one, and the ventral position in man be due to the strongly developed pyramidal fibres, which push out the olive.

2. At a point where the olive just disappears a second decussation of the pyramidal fibres takes place. The more laterally placed fibres become bent towards the middle line,

cross the mesial fibres, and decussate with those of the other side. A little higher the more mesially placed fibres also decussate.

The decussation is probably complete, and after decussation the pyramidal fibres become altered in position, those which were lateral become mesial, and *vice versa*.

This second decussation he cannot account for, but it is evidently peculiar to pteropus, for it was found in the brains of other three which were examined.

EDWIN MATTHEW.

**A CONTRIBUTION TO OUR KNOWLEDGE OF THE CORTICO-  
(169) BULBAR AND CORTICO - PONTINE PYRAMIDAL  
FIBRES IN THE HUMAN SUBJECT. RENÉ SAND, *Arch. a. d.*  
*Neurol. Inst. a. d. Wiener Univ.*, H. x., 1903, p. 185.**

FIVE human brains were examined. The lesions, cortical and sub-cortical, involved the fibres of the pyramidal tract, and were recent enough to allow of the Marchi method being employed in tracing the resulting degenerations.

In the mesencephalon the fibres of the pyramidal tract are found to occupy the middle third of the pes pedunculi. In the pons the cortico-bulbar and cortico-spinal fibres are intermixed. The cortico-bulbar fibres come off from all aspects of the pyramids, but chiefly from the mesial bundles. In the medulla oblongata the degeneration is most marked in the dorso-lateral angle of the pyramid; the fibres to the homolateral nuclei come off from this angle, whilst those to the contralateral nuclei leave the dorsal aspect and dorso-mesial angle.

Hoche and others believe that the cortico-bulbar fibres have a double path caudalwards—one through the fillet, the other by way of the pyramidal bundles. The author finds no fibres passing downwards by the fillet in any of his cases, although many, after leaving the pyramidal tract on their way to the motor nuclei, do pass obliquely through it.

The extra-pyramidal course of the cortico-pontine and cortico-bulbar fibres he found to be as follows:—No fibres are seen coming off in the region of the oculo-motor nucleus. At the level of the decussation of the trochlear nerve the first fibres leave the pyramid. These pass off in bundles from its dorso-lateral aspect and run to the middle segment of the fillet in which they disappear. At the junction of the upper and middle thirds of the pons numerous fibres run backwards, reaching the middle and lateral parts of the fillet, where they form small scattered bundles directed obliquely spinalwards. At the level of the motor nucleus of the fifth the fibres are isolated and no longer fasciculated. The more mesial cross the raphé and pass with the internal arcuate

fibres to the motor nucleus of the fifth of the opposite side; the lateral fibres pass through the fillet to the homolateral nucleus. In the lower levels of the pons and upper levels of the medulla oblongata isolated fibres run from the mesial aspect of the pyramid to the neighbourhood of the opposite facial nucleus, whilst similar fibres from the dorso-lateral edge pass to the nucleus of the same side. The fibres for the nuclei of the twelfth were only slightly degenerated, and their course could not be made out with exactness. The author has traced all these fibres to the neighbourhood of the above named motor nuclei, and he believes that they end in these nuclei.

In all the cases numerous degenerated fibres pass through the corpus callosum and are found in the pyramidal tract of the opposite side. In one case the fibres of the bundle named by Spiller the "direct ventro-lateral pyramidal tract" are degenerated, and the author thinks that the cerebral origin of these fibres is certain. Degeneration was also found in the superior cerebellar peduncle. These cerebrofugal fibres come either from the cortex or the corpus striatum, or both, as neither the optic thalamus nor red nucleus was injured in any of the cases in which they were found.

SUTHERLAND SIMPSON.

**A CONTRIBUTION TO THE MORPHOLOGY OF THE APES**

(170) **BRAIN.** E. ZUCKERKANDL, *Schwalbe's Ztschr. f. Morphol. und Anthropol.*, Bd. iv., 1902, p. 463.

**NOTE ON THE SO-CALLED "TRANSITORY FISSURES" OF**

(171) **THE HUMAN BRAIN, WITH SPECIAL REFERENCE TO BISCHOFF'S "FISSURA PERPENDICULARIS EXTERNA."**  
G. ELLIOT SMITH, *Anatomischer Anzeiger*, Bd. xxiv., 1903, p. 216.

**THE VARIATIONS IN THE CEREBRAL FISSURES IN APES,**

(172) **WITH SPECIAL REFERENCE TO THE "AFFENSPALTE"**  
J. H. F. KOHLBRUGGE, *Ztschr. f. Morphol. und Anthropol.*, Bd. vi., 1903, p. 191.

**A CONTRIBUTION TO THE MORPHOLOGY OF THE APES**

(173) **BRAIN (Zweiter Beitrag).** E. ZUCKERKANDL, *Ztschr. f. Morphol. und Anthropol.*, Bd. vi., 1903, p. 285.

**THE MORPHOLOGY OF THE RETRO-CALCARINE REGION OF**

(174) **THE CORTEX CEREBRI.** G. ELLIOT SMITH, *Proceedings of the Royal Society*, Vol. lxxiii., 1904, p. 59.

**THE COMPARATIVE ANATOMY OF THE OCCIPITAL LOBES.**

(175) E. ZUCKERKANDL, *Abh. a. d. Neurol. Inst. a. d. Wiener Universität*, H. 10, 1904, p. 297.

**THE MORPHOLOGY OF THE OCCIPITAL REGION OF CEREBRAL HEMISPHERE IN MAN AND THE APES.** G. ELLIOT SMITH, *Anatom. Anzeig.*, Bd. xxiv., 1904, p. 436.

**STUDIES IN THE MORPHOLOGY OF THE HUMAN BRAIN, WITH SPECIAL REFERENCE TO THAT OF THE EGYPTIANS—NO. 1, THE OCCIPITAL REGION.** G. ELLIOT SMITH, *Records of the Egyptian Government School of Medicine*, Vol. ii., 1904.

It is perhaps not an exaggeration to say that no problem in cerebral morphology has given rise to more controversy (with, let me add, so little positive result in the way of agreement in regard to the main point at issue) than that which has resulted from the discussion of the resemblances and differences in the occipital region of the hemisphere in man and the apes. It was the subject of one of the most notorious of the many disputes which the publication of Darwin's "Origin of Species" in 1859 aroused in the following decade; and although "the subject matter of the dispute is now totally different from what it was formerly," as Huxley remarked in reference to this discussion in 1874 (Darwin's "Descent of Man," 2nd edition), "the controversy has not yet come to an end." In fact, the thirty years which have elapsed since these words of Huxley's were published, have only added more force to his statement, for hardly a year has elapsed since then without adding appreciably to the vast mass of literature dealing with the supposed distinctive features of the occipital region of the human brain. The 108 memoirs quoted by Pfister in 1899 ("Über die Occipitale Region," Stuttgart) by no means exhausts the list, for many well-known monographs, such as that of Sir William Turner, do not figure in his enumeration.

It is no mere figure of speech to describe the results of all this research as chaotic. There is the greatest possible disagreement between the conclusions of these writers and the most recent memoirs of Zuckerkandl and Kohlbrugge, and the writer adds still further to this divergence of view.

Most recent writers, such as Cunningham, Pfister, Zuckerkandl and Kohlbrugge, believe that there is no furrow in the normal adult human brain which can be regarded as the homologue of that called "Affenspalte" by Rüdinger in the apes. "There is not a trace of such an arrangement in the human brain" (Cunningham, *British Association Reports*, 1901).

But last year I found that a considerable proportion of the simple African brains possessed a furrow (which I called *sulcus lunatus*, in reference to its commonest shape) obviously identical in every respect with the sulcus in the gorilla, which most writers (and certainly Cunningham and Zuckerkandl) would not hesitate

to call "Affenspalte" (see the abstract in this *Review*, Vol. ii. No. 1, p. 55). Subsequently I discovered that the sulcus lunatus occurred in other, including the European races, and hence came to the conclusion that Bischoff and Cunningham could not be correct in their belief that the "Affenspalte" developed in the foetal brain ("fissura perpendicularis externa") and subsequently became obliterated. Further enquiry showed that their supposed "Affenspalte" in the human foetal brain was merely an indentation produced after death by the inward bulging of the membrane in the lambdoid suture (*Anat. Anz.*, 1903, p. 216).

Subsequently I discovered a very simple means of settling once for all the much discussed problem of the means of identifying the "Affenspalte" in the human brain. Nature has provided us in the streak of Gennari a very clear and certain guide to the situation of the sulcus lunatus or "Affenspalte" (*Proc. Roy. Soc.*, Jan. 28th, 1904, p. 59). It affords us evidence which requires none of the refinements of modern technique for its demonstration; for it is now 128 years since Gennari described the white line in the occipital cortex. Much labour has recently been bestowed on the study of the detailed structure of Gennari's stria, which Ramon y Cajal and Henschen have demonstrated to be formed largely of the optic radiations of Gratiolet; it has, moreover, been known for many years—I think Munk was the first to record the fact—that the white streak in question stops abruptly in the free edge of the occipital operculum in the Macaque, *i.e.* the caudal lip of the "Affenspalte"; and yet no one, so far as I am aware, had attempted to correlate these facts and make the obvious comparison of the distribution of the area striata (the cortical region containing Gennari's stria) in man and the apes with the view to determine the position of the "Affenspalte" until my preliminary notes were published in the *Proceedings of the Royal Society* and the *Anatomischer Anzeiger* (1904, p. 436). In a considerable proportion of human brains the streak of Gennari extends outward on to the lateral aspect of the hemisphere and stops abruptly in the anterior free edge of the operculum, which a study of the mere superficial form had led me to regard as the caudal lip of a genuine homologue of the "Affenspalte." *This finally establishes beyond all question whatsoever the identity of the human and the anthropoid sulcus lunatus or "Affenspalte";* but if more evidence is wanted, it is easy to select human brains in which the whole pattern of the laterally-placed occipital sulci of the apes—sulcus lunatus, the Y-shaped sulcus occipitalis superior (intrastratus, mihi) and sulcus occipitalis inferior (infrastratus, mihi)—is exactly reproduced. But the abrupt anterior edge of Gennari's streak is the real criterion.

The sulcus lunatus is, in fact, a depression produced by the



forward projection of the anterior edge of the stria-bearing area. It may be a deep cleft undermining the operculum, the superficial layer of which consists of the area striata; it may be a simple sulcus; or it may be a shallow pucker crossed by irregular bridging gyri. In its most pronounced form the operculum projects forward to overlap the caudal end of the intraparietal sulcus and the incisura parieto-occipitalis. But these two sulci have no more right to be regarded as constituent elements of an "Affenspalte," as Kohlbrugge and others suppose, than the river Rhine has to be called part of the German Ocean. The whole cleft is due to the tongue-like, forward projection of the cortical area containing the streak of Gennari; whether or not its caudal opercular wall overlaps any other sulci is a matter of the very slightest importance. But if this overlapping is to be considered a criterion of the "Affenspalte" it does not much matter, seeing that this term is already so inappropriate and misleading that a mere verbal quibble as to its exact meaning is a perfectly innocuous occupation. But the pucker (or sulcus, or cleft, whichever it may be) which is produced by the forward projection of the anterior edge of the area striata is a feature—the sulcus lunatus—of definite morphological value, which is present in many New World apes, all Old World apes, and the vast majority of human beings of probably every race. It is the furrow which most writers call "Affenspalte"; and when Kohlbrugge, without adducing any evidence in support of his statements, says: "Will man nun  $m + m^1$  [sulcus lunatus, mihi] als Affenspalte bezeichnen, dann würden alle Anthropoiden und auch der Mensch immer eine Affenspalte besitzen, denn  $m$  fehlt nie" (p. 242), he assumes as a fact, what a host of anatomists have been disputing for more than fifty years. If the sulcus  $m$  is the "Affenspalte," the latter is present, as I have shown (but Kohlbrugge has certainly *not* done so), in the human as well as in the ape brain; but if the term "Affenspalte" can only be properly applied to the cleft-like lunate sulcus when its caudal lip overlaps the parieto-occipital sulcus, it can only be said to exist in a limited number of apes: it thus ceases to be a distinctive feature of the apes contrasted with man, and becomes merely a useless character differentiating certain *individuals* of some genera of apes from all the other Primates. It seems to me that Kohlbrugge's contention is either an utterly futile, though *perhaps* (?) academically correct view, or a mere verbal quibble.

During the last three years Zuckerkandl has published three memoirs which are devoted almost wholly to the comparison of the occipital region of the hemisphere in various Primates.

His first memoir deals with all the cerebral sulci of the Primates, more especially those of the Woolly-Monkey (*Lagothrix*), the Spider-Monkey (*Ateles*) and the Gibbon (*Hylobates*). The chief

subject discussed in this memoir is the nature of the "Affenspalte" and the annectant gyri. Zuckerkandl, following closely in the footsteps of Gratiolet and Sir William Turner, describes three external gyri bridging the parieto-occipital boundary line.

His second memoir deals exclusively with the morphology of the parieto-occipital fossa and the lateral annectant gyri. He gives most excellent representations of these gyri in various Cercopithecidae (Taf. xi.). It is, however, his third memoir which is more especially concerned with the discussion of the lateral occipital region and the "Affenspalte." He begins with a detailed analysis of the claims of all the various sulci in the human brain which different writers have regarded as the "Affenspalte." Bischoff's sulcus perpendicularis externus, Ecker's sulcus occipitalis transversus, Wernicke's sulcus occipitalis anterior, Meynert's sulcus occipitalis externus are in turn examined and their right to be regarded as the ape-fissure itself or as part of it is utterly disproved. He traces the evolution of the sulcus occipitalis anterior of Wernicke—he calls it sulcus gyri angularis—from the New World apes through the Old World apes up to man, and shows that it is a relatively primitive furrow placed far in front of the "Affenspalte" in all apes, so that the anterior occipital sulcus in the human brain cannot be the "Affenspalte"; and *a fortiori* Meynert's sulcus occipitalis externus, which is placed still further forward, is put altogether out of count.

When we leave the critical, analytic part of Zuckerkandl's work and pass to the statement and discussion of his own views in regard to the "Affenspalte," his argument becomes extremely difficult to follow. At the outset, I may state that the whole of his comparisons of the human and the ape brain are open to the serious, if not fatal, objection that instead of comparing the human brain with those of the anthropoid and Old World apes, which are linked by the closest bonds of affinity to man and exhibit the nearest structural resemblance to him, he has attempted to force a comparison with the most specialised New World apes, which have developed along lines entirely divergent from that common to man and the Simiidae. The result of this strange procedure is to utterly stultify (so far as the search for the representative of the "Affenspalte" in the human brain is concerned) the vast amount of patient research which his three memoirs must have entailed.

The preoccupation which this defiance of the teaching of zoology has entailed can be the only explanation of his failure to note the obvious identity of the typical sulcus lunatus ("Affenspalte") which one of his human specimens (third memoir, figure 25, tr.<sup>2</sup>) exhibits and the "Affenspalte" of his chimpanzees (see his figure 9, for example) and oranges (figures 4, 5 and 6).

The explanation of this procedure seems to be that the New World apes exhibit the most primitive forms of the "Affenspalte," and the occipital operculum has not advanced in them sufficiently far to cover the annectant gyri. Hence Zuckerkandl has selected the brains of the New World apes to compare with that of man, because in him the annectant gyri are exposed and the "Affenspalte" presumably small. The further great fallacy of his whole argument is that he accords to the three annectant gyri a morphological individuality which they certainly do *not* possess. The annectant gyri are the results of the fortuitous breaking up of the cerebral surface by a group of most unstable sulci, such as the sulcus occipitalis transversus and the rest of the caudal part of the intraparietal sulcus, the course and form of which are determined by purely mechanical conditions, exhibiting the utmost variability in different Primates.

In its most typical form, as seen in a Macaque or a Cercopithecus, the "Affenspalte" (see Z.'s first and second memoirs) is a great cleft in which are submerged the first annectant gyrus (between the incisura parieto-occipitalis and the caudal end of the intraparietal sulcus), the second annectant gyrus (external to the intraparietal sulcus), and the third annectant gyrus, separated from the second gyrus by an inconstant furrow, which I believe to be devoid of any morphological value. The "Affenspalte" of *Ateles* (third memoir, figures 1, 2, 21 and 22) is a furrow which fulfils all my criteria of a sulcus lunatus, i.e. it is the depression formed at the interior margin of the area striata by the forward bulging of the latter. But according to Zuckerkandl, only the extreme anterior (lower) end can be considered as "Affenspalte"; the rest is the "Furche zwischen 2 und 3 Übergangwindung einerseits und dem Hinterhauptlappen anderseits": if I understand it rightly this statement means that the upper (posterior) part of the sulcus lunatus of *Ateles* is not "Affenspalte" but the bottom of the "Affenspalte"—a distinction, the force of which is not altogether apparent, especially when it is recalled that the relation of the stripe of Gennari to the caudal lip of the furrow is identical with that found in the Macaque, and, in fact, in all Primates. But the inference drawn from this statement is more extraordinary still: the "Furche zwischen 2 und 3 Übergangwindung und dem Hinterhauptlappen" is said to be homologous to the lateral occipital sulcus of the human brain (p. 349): this leads up to the *reductio ad absurdum* of his work, which I must quote in his own words: "Wenn ein Rest der Affenspalte noch zur Entwicklung käme, so müsste derselbe an das VORDERE [mihi] Ende der lateralen Occipitalfurche angeschlossen sein, was aber an meinem Material nicht zu erkennen ist" (p. 350). This means that he believes that no "Affenspalte" is present in the human brain, but if it should happen to persist it would be placed

at the *anterior* end of the lateral occipital sulcus—which, in the sense implied in Zuckerkandl's memoir, is probably my "sulcus prælunatus." His imaginary human "Affenspalte" is therefore a sulcus "præ-prælunatus"! *i.e.* in front of the sulcus which I have already described as being in front of the real "Affenspalte"! But my evidence is quite conclusive in regard to the *facts* of the case, however much one may quibble as to the meaning of terms. The sulcus lunatus, which is identical with what most writers call "Affenspalte," is certainly caused by the forward projection of a cortical area containing the streak of Gennari. This is the real causal factor in the production of the "Affenspalte," both in the apes and in man; and the question whether the opercular caudal lip covers a small or a large cortical area is of quite subsidiary importance.

G. ELLIOT SMITH.

**ON THE COMPARATIVE ANATOMY OF THE ASSOCIATION  
(178) SYSTEMS OF MAMMALIAN BRAINS—I. THE CINGULUM.**

E. REDLICH, *Arch. a. d. Neurol. Inst. a. d. Wiener Universität*  
(H. Obersteiner), H. x., 1904, p. 104.

THIS paper is the first instalment of a pretentious plan, to study the comparative anatomy of the association-tracts of the mammalian forebrain.

It is evidently impossible to extend the work to the nervous system of the lower vertebrates, and difficult to attempt to homologise even the longer and more important bundles of the forebrain of the marsupials with those of the placental mammals, owing especially to the absence of a corpus callosum.

The author devotes the paper under notice to the study of one of the best known of these forebrain tracts—the cingulum—whose relations and connections he has studied in representatives of nearly every class of the placental mammals.

The most important conclusions of the work are that the cingulum does not contain projection fibres, and that it undoubtedly has definite relations to the olfactory centres. Its size stands in direct relation to the development of the latter, it is large in macrosomatic and small in microsomatic animals.

The stria Lancisii medialis is an analogous structure to the cingulum and should be regarded as part of it. The fibres of the cingulum are not limited to the limbic convolution, but also extend into the marginal gyrus, so that they are not merely in connection with the former, but with the whole medial surface of the hemisphere dorsal to the corpus callosum.

In all placental mammals, fibres from the cingulum and from the stria Lancisii pierce the corpus callosum to form the fornix

longus. This, which passes basalwards in the septum pellucidum to the olfactory area, represents an important part of the cingulum. The rest of it runs forwards parallel to the corpus callosum, and at its genu sends fibres into the frontal lobe, while the remaining bend ventralwards into the septum pellucidum to end, in macrosmatic animals at least, in the grey matter round the ventriculus olfactorius. As it posteriorly bends round the splenium, some fibres are similarly given off to the occipital lobe and the remaining pass forwards and basalwards to end in the alveus of the cornu Ammonis.

Thus at least two different groups of fibres can be distinguished in the cingulum: a group which runs parallel to the corpus callosum in the dorso-medial wall of the hemisphere containing association-fibres between its various portions; and a second which runs more or less vertical to the corpus callosum and connects the dorso-medial wall of the hemisphere with the ventro-medial (olfactory) portion. Included in this group are the fibres of the fornix longus. Though Redlich has been unable to definitely prove it, the evidence he brings tends to show that the latter group conduct from the basal olfactory centres towards the medial cortex.

The cingulum is thus not a simple association-tract of the medial wall of the hemisphere, but a complex system inserted in the central olfactory system, which contains fibres of different length and of various origin and destination.

GORDON HOLMES.

**THE SPINAL CORD OF THE PORPOISE (PHOCOENA COM-  
(179) MUNIS).** HEPBURN and WATERSTON, *Journ. of Anat. and  
Phys.*, Vol. xxxviii, Jan. 1904, and April 1904.

THE authors describe the structure of the spinal cord of the porpoise, and compare the conditions found in it with those found in other mammalian cords.

The chief points elucidated are, that there is a well-marked lumbar enlargement, notwithstanding the fact that the hind limbs are practically entirely absent, but that the grey matter of the cord in this region does not show the lateral enlargement found in that region in man.

Sections were examined from every segment of the cord, and it was found that the shape of the grey matter was distinctive for each segment.

The superficial area of the grey matter and of each of the columns of white matter was also determined, and the results show that in this mammal the area of all the columns is markedly

increased in the region of the enlargements, indicating the appearance of numerous short commissural fibres at those levels in all the columns.

The alterations in the shape of the grey matter from segment to segment, associated with the appearance of groups of motor nerve cells, are analysed and compared with those in man, and the results generally comprise our knowledge of the position of the nucleus for the phrenic nerve, for the perineal muscles, and for the muscles of the limbs and trunk.

A heterotopia of the grey matter in the posterior columns is also described, and has since been confirmed by the work of Guldberg on the same lines.

It was also found that the nucleus of the spinal accessory nerve appeared to be double, one nucleus being situated near the antero-lateral aspect of the anterior horn, while a more posteriorly placed nucleus was found near the base of the anterior horn.

The plates show photographs of typical sections in every cervical segment, and in the more striking of the dorsal and lumbo-sacral series.

DAVID WATERSTON.

**THE DEGENERATIONS RESULTING FROM LESIONS OF THE (180) POSTERIOR NERVE ROOTS AND FROM TRANSVERSE LESIONS OF THE SPINAL CORD IN MAN.** A study of twenty cases. JAMES COLLIER and E. FARQUHAR BUZZARD, *Brain*, No. 104, Vol. xxvi., 1904, p. 559.

Two cases of lesion of the lumbo-sacral roots were examined by the Marchi method (Busch's modification). In one case the third lumbar posterior root of the right side was alone degenerated. In the other case all the posterior roots below and including the second lumbar were degenerated except the right third lumbar posterior root. Both were cases of new growth involving the spinal column. The degenerations are described in detail and figured by projection drawings and photographs. The ascending degenerations corresponded with the generally accepted description. Descending degeneration was traced from the degenerate roots: (1) into the ventral field of the posterior column of the same side; (2) inclining across the posterior column to reach the side of the median septum where the degenerate film occupied the oval area of Flechsig and were continued into the sacral triangle, and in rapidly decreasing numbers were traceable as far as the grey matter of the conus medullaris.

The following conclusions are arrived at:—

- (1) Descending collaterals are given off in large numbers from the lumbar posterior roots and end in the grey matter of

the cornu-commissural region, and in the grey matter of the dorsal horn of the same side. They vary in length, the longest reaching through many segments, and these are traceable as far as the grey matter of the conus medullaris.

- (2) The oval area and sacral triangle contain exogenous fibres as well as fibres continuous with the bandalette of Hoche. There is no definite evidence at present as to whether the latter fibres are endogenous or exogenous in their origin.
- (3) The ventral field of the posterior column contains some exogenous fibres, and these are most numerous towards the median part of the ventral field.
- (4) In the lumbo-sacral region a small triangular area lying superficially and mesial to the entering posterior root is composed almost entirely of endogenous fibres.

The cases of transverse lesions of the cords were cases of traumatic paraplegia, pressure paraplegia, and acute myelitis, in which the damage to the spinal cord was strictly local. Twelve of these were examined by the Marchi method. The degenerations found are figured with projection drainings and photographs, and a schematic diagram of the direct cerebellar tract and of Gower's tract, etc., is appended.

*Descending Degenerations.*—The termination of fibres of the pyramidal tract in the region of Clarke's nucleus, and the absence of any fibres passing from the region of the lateral pyramidal tract to any part of the ventral horns, is demonstrated. An examination of the lateral limiting layer of the spinal cord immediately above and below lesions at various levels failed to corroborate the hypothesis that the ventral part of this region is made up of descending fibres of short intraspinal course, and that the dorsal part is composed of ascending short intraspinal fibres.

*Descending Degeneration in the Posterior Columns.*—This was found to be remarkably constant in its course and distribution.

Immediately below a lesion situated at any level above the tenth dorsal segment, the chief descending degeneration in the posterior column took the form of a strip stretching across the postero-external column, from the junction of the dorsal horn and dorsal commissure to a point on the surface of the cord some distance mesial to the entrance of the dorsal root. The large fibres of this degeneration tended to collect at the ventral end and at the dorsal end of this strip, and for this reason the area of degeneration was somewhat dumb-bell shaped.

Whatever the level of the lesion, the descending strip of degeneration in the posterior column became separated into a ventral

portion and a dorsal portion at the twelfth dorsal segment. The ventral division (sulco-commissure bundle of Dufour) disappeared constantly in the fifth lumbar segment, its fibres entering the grey matter of the cornu-commissural region. Some of the coarse fibres which it contains passed to the septum, and running dorsally, entered the oval area in the third lumbar segment. The dorsal division, composed of large fibres, formed the peripheral bandalette of Hoche in the twelfth dorsal segment, and the fibres reaching the septum in the first and second lumbar segment occupied the oval area in the lower three lumbar segments, and forming the sacral triangle in the third sacral segment entered the posterior grey matter of the same side in the lowest sacral and coccygeal segments. So far the description of the dorsal division corresponds with that of Hoche and subsequent writers, but in addition, fine fibres are described which remain throughout their course in the dorso-external region of the posterior columns, and which were traced in lesions of the mid-dorsal region downwards as far as the conus, where they course ventrally through the thickness of the posterior columns to enter the grey matter.

Special attention is drawn to the anatomical limitation of the oval area and sacral triangle by connective tissue septa.

Hoche's ventral septal fibres were found to be constantly present, though in variable numbers. In lesions of the cervical and upper dorsal regions they were traceable through two segments, but in lesions at lower levels they could not be followed for more than one segment; their termination was in the posterior grey matter, which they reached by passing ventrally along the septum.

*Ascending Degeneration.*—Some fibres of the dorsal longitudinal bundle were degenerated above the lesions, and the number of these were greater the higher the level of the lesion in the spinal cord. Only a few fibres could be traced above the sixth nucleus.

The ascending degenerations in the posterior columns were found to coincide with the classic description. No fibres were traced in any of the cases from the posterior columns to the cerebellum, neither as external nor as internal arcuate fibres, but degenerate fibres could be traced from the region of these nuclei into the grey matter lying mesial to the restiform body, as have been demonstrated by Horsley and Thiele.

*The Direct Cerebellar Tract.*—This system of fibres is described in considerable detail, and the following points are emphasised:—

- (1) The fibres of this system can be distinguished from other ascending degenerate fibres in the lateral column by their greater size.

- (2) The bulk of the fibres have their origin in the lumbar and cervical enlargements of the spinal cord.



(3) The most dorsally placed fibres of the tract constitute a very definite spino-vestibular system. They are anatomically separated in the lower medulla from the other fibres of the tract as a tongue-shaped area of degeneration extending backwards from the direct cerebellar tract and lying between the substantia gelatinosa and the surface of the cord. They are the first to pass away as external arcuate fibres, and running in the thin grey layer covering the restiform body pass over the dorsal aspect of that structure and sink ventrally, breaking up in the grey matter around the descending vestibular root.

(4) The ventral and dorsal medullary plexuses were constantly present and were formed by collaterals of the fibres of the direct cerebellar tract.

(5) Fibres were traced to the peduncle of the flocculus in every case, and many fibres entered the fleece of the dentate nucleus.

*Gowers' Tract and the Associated Spino-tectal and Spino-thalamic Tracts.*—Fibres of Gowers' tract were traced: (1) to the direct cerebellar tract; (2) to the middle peduncle of the cerebellum; (3) to Bechterew's nucleus and to Deiters' nucleus; (4) ascending with the descending root of the fifth nerve; (5) to both posterior colliculi, *via* the velum; (6) to the superior vermis. It is further suggested that fibres of this tract may enter the collateral medullary plexuses.

The spino-tectal fibres were followed to the nucleus of the lateral fillet and to both colliculi of either side.

The spino-thalamic tract was not traceable further than the ventral nucleus of the thalamus. Just as these fibres take up their characteristic position ventral to the mesial geniculate body, some fibres left the tract to end in the lateral portion of the substantia nigra.

AUTHOR'S ABSTRACT.

## PHYSIOLOGY.

### ON THE CENTRIFUGAL FUNCTION OF THE POSTERIOR ROOTS (181) OF THE SPINAL CORD. CESARE POLI, *Ann. di Freniatria*, Vol. xiii. F. 4, and Vol. xiv. F. 1, 1904.

THE presence of centrifugal fibres in the posterior roots of the spinal cord was first established by Ramon y Cajal and Lenhossek, the latter of whom stated that they took their origin from the posterior root cells of the anterior grey matter of the cord, and that they passed through the posterior root ganglia without forming any connections in them.

The existence of these fibres in the posterior roots has been denied by many, but it has been placed on a firm basis by physio-

logists, among whom Stricker and Bayliss take a prominent place.

Stricker, in 1876, stated that "the sciatic nerve in the dog receives vaso-dilator fibres directly from the lumbar posterior roots, and that these fibres do not go through the sympathetic system."

Bayliss has come to the following conclusions:—

1. The vaso-dilator nerves of the posterior limb of the dog run in the posterior roots of the 5th, 6th and 7th lumbar and 1st sacral nerves, and none pass in the anterior roots.

2. The vaso-dilator nerves of the anterior limb run in the posterior roots of the 6th, 7th and 8th cervical and 1st dorsal nerves.

This vaso-dilator action still takes place after the lumbar spinal cord has been removed, but stimulation of the peripheral end of a cut sciatic nerve with an infrequent electrical current does not produce a vaso-dilator action if the posterior root ganglia, connected with the roots of this nerve, have been destroyed nine days before the experiment is made.

This proves that the centres for this vaso-dilator action are outside the cord, and that they lie in the posterior root ganglia.

To explain the vaso-dilator action of these posterior root fibres, Bayliss suggests that efferent stimuli pass along the afferent nerves. It is possible, however, that one of the five types of cells found in the posterior root ganglia (Dogiel) may have a vaso-dilator function. On the other hand, Rattone has described cellular elements in the posterior roots of mammals, and these may have a vaso-dilator action.

In 1882, Mosso and Pellacani stated that the nerve fibres which control the action of the bladder descend in the posterior columns and in the extreme posterior part of the lateral columns of the cord, and that motor fibres for the bladder do not exist in the anterior and antero-lateral columns.

It is probable, therefore, that they pass out of the cord by the posterior roots.

In order to investigate this point the author of this paper began a series of experiments on dogs, but his work was unfortunately interrupted, and we have here just a short account of what he found in five dogs experimented on. The nerves which innervate the bladder are the erector nerve of Eckhard and the hypogastric nerve. Centres of control are found in the cerebrum, the spinal cord, and the sympathetic system; but it must also be admitted that the bladder can functionate normally by means of the co-ordinated reflexes of the intra-vesicular ganglionic plexuses.

The results of these experiments were inconstant, and in the different cases contraction of the bladder followed stimulation, sometimes of one root and sometimes of another. In the first three cases contraction of the bladder was obtained by stimu-

lating the 2nd and 3rd lumbar roots, and in the last two cases by stimulating the 4th and 5th roots. Once it followed stimulation of the 6th root.

The author, after having carefully discussed any sources of error which might have interfered with his results, states that :—

1. Some posterior roots in the lumbar region of the dog, stimulated electrically or mechanically, produce a contraction of the bladder.

2. The contractions obtained have a duration longer than that of the stimulus applied, and are more or less marked according to the condition of the animal and of the nerve.

3. In the separate contractions he was always able to distinguish a latent period, which also varied with the above-mentioned conditions.

R. G. ROWS.

**ON THE UNION OF THE FIFTH CERVICAL NERVE WITH  
(182) THE SUPERIOR CERVICAL GANGLION. J. N. LANGLEY  
and H. K. ANDERSON, *Journ. of Physiol.*, Feb. 1904, p. 439.**

THE authors in the case of two young cats removed  $2\frac{1}{2}$  cm. of the right cervical sympathetic. The fifth cervical nerve where it issues from the muscles ventrally was then cut, and its central end united to the peripheral end of the sympathetic. Several months afterwards the animals were anaesthetised and the nerves stimulated, and it was found that in both cases stimulation of the fifth cervical nerve caused, in a greater or less degree, the usual effects produced by stimulation of the cervical sympathetic.

Langley had previously shown that the pre-ganglionic fibres of the vagus are capable of becoming functionally connected with the nerve cells of the superior cervical ganglion. The fifth cervical nerve contains no pre-ganglionic fibres; it contains efferent and afferent somatic fibres and post-ganglionic sympathetic fibres, and it was in order to find out whether any of these classes of fibres could make functional connection with sympathetic nerve cells that these two experiments were made.

Nicotine injected intravenously or applied locally to the superior cervical ganglion abolished, for a time, the normal effect of stimulating the cervical sympathetic. This showed that some fibres of the fifth cervical nerve had become functionally connected with nerve cells in the superior cervical ganglion. These, the authors conclude, are probably efferent somatic fibres, and they are carrying on further observations in order to determine whether "this power belongs—as we should naturally assume—to efferent fibres only, and, if so, whether it is universal or confined to certain classes of efferent fibres."

The sympathetic nerve between the point of junction and the superior cervical ganglion was examined microscopically. In both cases it consisted almost entirely of medullated nerve fibres, a considerable number of which were larger than those found normally in the cervical sympathetic. "The occurrence of these fibres appears to be in favour of the view that in regeneration the new fibres grow out from the central nerve stump, and to be against the view that such fibres are formed *in situ* in the peripheral cut portion."

SUTHERLAND SIMPSON.

## PSYCHOLOGY.

**THE METHODS OF MEASURING FATIGUE IN SCHOOL**  
(183) **CHILDREN.** By M.-C. SCHUYTEN, *Arch. de Psychol.*, T. ii., Oct. 1903, p. 321.

IN this paper the author criticises the various endeavours which have been made to estimate the fatigue of school children by testing their auditory memory for figures at different hours of the day. These observations have generally seemed to indicate that fatigue is greater in the afternoon than in the forenoon. The writer finds that when the test is first applied in the afternoon, and repeated on the following forenoon, a contrary result is obtained, and fatigue appears to be greater in the morning. The explanation of this discrepancy the author takes to be that the first time the test is applied the children are interested in what is being done, but on the second occasion the novelty of the proceeding has worn off, and accordingly they are less attentive, and so the memory appears to be less active. The conclusion to be drawn is, that the test, as usually applied, is worthless as a measure of fatigue. If it is to be used, a fresh set of children (of the same age, intelligence, etc.) should be taken for each observation. Incidentally, the author finds that the auditory memory for figures is better in girls than in boys.

W. B. DRUMMOND.

## PATHOLOGY.

**THE BRAIN IN A CASE OF CONGENITAL BILATERAL ANOPH-**  
(184) **THALMUS.** VICTOR HANKE, *Arch. a. d. Neurol. Inst. a. d. Wiener Univ.*, H. x., 1903, p. 58.

PROFESSOR HANKE describes in his paper the condition of the brain of a child with bilateral anophthalmus. The child lived eight days after birth, dying of bronchitis. Eyelids were present, the orbital fissure was very narrow, and a blind sac lined with mucous membrane formed the orbit. No movement of the eye was visible while the child lived, but microscopically a small piece of pig-

mented tissue, the size of a poppy seed, embedded in fat, was found in the sac.

Round this mass were the eye muscles, all present, and each had its proper nerve supply.

The mass consisted of rudimentary sclerotic and choroid. In the brain there was no trace of an optic chiasma or optic tract.

Microscopically, H. examined the external geniculate body, the anterior corpora quadrigemina, and the occipital lobe in the region of the calcarine fissure.

The external geniculate body was reduced in size, being only as large as a hemp seed. On section the ganglionic cells were found greatly diminished in numbers and in size, more especially at the peripheral part of the ganglion. The anterior corpora quadrigemina showed no change in form. The stratum zonale was absent and in the grey matter the cells were again scanty in number and deficient in size.

In the occipital lobe, the thickness of the cortex was diminished in a marked degree when compared with that of a normal child, in the proportion of .884 mm. to 1.456 mm. The fourth layer was entirely wanting, and layers one and two were diminished in thickness. Here also the cells were less numerous, and those present were smaller than usual.

Hanke considers his case brings out two very important facts:—

1. The absence of the stratum zonale in the anterior corpora quadrigemina.
2. The changes found in the layers of the occipital lobe.

He then proceeds to analyse the various written opinions as to the function of the stratum zonale in connection with sight. Some authorities consider it contains sight fibres, and some think it does not. This case indicates that the first view is the correct one. Again, in cases where the organs of sight are absent, either congenitally or experimentally in animals, various findings have been recorded as to the condition of the occipital lobe. H. records the various views, and considers that those of Leonowa come nearest to his case; and he considers that, as in the brain he examined, in a total absence of the fourth layer in the occipital cortex, the organs of sight cannot possibly be developed. EDWIN MATTHEW.

**THE CENTRAL NERVOUS SYSTEM IN CRETINS.** BAYON, (185) *Neurolog. Centralbl.*, No. 8, 1904.

THE writer here gives an account of the conditions present in the brains of two cretins, of whom one was 86 years old and the second 25.

The second brain was immersed in 10 per cent. formalin one and a half hours after death, and as the patient died from acute cardiac failure, there was no "late infection" during life and no post-mortem change.

The changes in the old brain consisted in the presence of a large number of amyloid bodies and thickening of the vessels, while the nerve cells showed practically no change.

In the young specimen there were practically no changes of either kind—no amyloid bodies, vessels were thin-walled, and the cells also were normal in appearance, there being no alteration in the cell body or in the processes.

Comparing the clinical history and the post-mortem appearances, the author concludes that the first case shows results of a "cachexia thyreopriva," with marked senile changes; while the latter affords a good example of cretin infantilism.

DAVID WATERSTON.

**THE PATHOLOGICAL ANATOMY OF TABETIC COMBINED (186) SCLEROSIS.** O. CROUZON, *Nouv. Icon. de la Salpêtrière*, 1904, p. 52.

THESE cases can be recognised clinically by the occurrence of dragging of the feet in walking and by the presence of the extensor type of plantar reflex, in addition to the usual symptoms of tabes.

The writer has made a detailed histological examination of six cases.

In some of these cases the lesions have been strictly limited to and determined by the fibre systems. In other cases the degenerations did not really correspond with the fibre systems, but their distribution was determined by lesion of the blood-vessels, the lymphatics and of the meninges.

Excellent illustrations of the conditions found in the spinal cord accompany this paper.

JAMES COLLIER.

**PATHOLOGICAL ANATOMY OF ACUTE DELIRIUM (ACUTE (187) DIFFUSE PARENCHYMATOUS CEPHALOPATHY).** ALESSI, *Riforma Med.*, No. 17, 1904.

THE material on which the author's remarks are based was obtained from a case of acute delirium. Prior to death, cultures were made on agar-agar and peptone gelatine from the blood. On the same media cultures were made from the longitudinal sinus and heart at the necropsy. All yielded a common pyogenic

coccus. Pieces from the cerebrum and cerebellum were fixed, stained and examined.

The protoplasmic processes of the large pyramidal cells stained faintly and showed chromatolysis at their bases. The cell body was deformed and showed a marked degree of chromatolysis with displacement of the nucleus and sometimes vacuolation. Some cells had a reticulated appearance, and around most altered cells there were collections of leucocytes. The nucleus was usually shrunken and the pericellular spaces dilated. By Weigert's method some fibres, especially in the tangential layer of Exner, were slightly swollen and nodose.

In the cerebellum the cell lesions were less marked. There was peripheral chromatolysis and slight dilation of the pericellular spaces. The nuclei in the basal ganglia and in the medulla showed the same changes. The vessels and neuroglia were not altered.

Allessi is of opinion that the lesion is a purely parenchymatous one and not at all of an inflammatory nature, and suggests that instead of acute delirium the term acute diffuse parenchymatous cephalopathy should be substituted.

DAVID ORR.

## CLINICAL NEUROLOGY.

**A NEW SIGN OF BASILAR MENINGITIS.** SQUIRES, *Med. Rec.*, (188) March 26, 1904, p. 496.

THE new sign, which the writer has invariably found in tuberculous basilar meningitis, even as early as the fourth or fifth day of the disease, consists in a rhythmical contraction and dilatation of the pupils. It is elicited in the following manner:—Grasp the sides of the head and produce forcible extension on the spinal column; as this is done the pupils will dilate, and the greater the extension the more extreme will the dilatation be. Upon flexion of the head the pupils contract, so that when the chin is brought close to the manubrium they are of minimal size. This can be done several times a minute, and each time the pupillary phenomenon will be repeated.

J. S. FOWLER.

**HÆMORRHAGIC FIBRINOUS MENINGITIS: SPASMODIC PARALYSIS: PLEGIA: LUMBAR PUNCTURES: MERCURIAL TREATMENT: CURE.** J. BABINSKI, *Bull. et Mém. de la Soc. Méd. des Hôp. de Paris*, Oct. 23, 1903.

BABINSKI gives the case of a woman who was suddenly seized without any apparent cause with pains in the dorso-lumbar region,

weakness in the inferior extremities, numbness of the left leg. For ten days she was able to get up and walk; about the eleventh day, as she was trying to get up, her limbs suddenly gave way, she fell and could not raise herself. Since then spasticity of inferior extremities, retention of urine. October 1902, entered hospital; for four months paraplegia and retention of urine persisted, incontinence of faeces, generalised pains most severe in the lower half of the body; cystitis. For some days the upper limb was numb: this disappeared. Feb. 9th, 1902, enters Babinski's service; inferior extremities spastic, knee and Achilles reflexes exaggerated, on the left side spinal epilepsy in faint degree; Babinski's sign present on both sides; abdominal reflexes lost; over lower half of body slight diminution of sensibility; left pupil larger than right; Argyll-Robertson phenomenon on left side. Mercurial treatment is begun. Feb. 13th, in *statu quo*. Lumbar puncture; fluid issues in a jet; 12 c.c. taken, greenish tint, forming firm coagulum, containing lymphocytes. Feb. 14th, patient complained on the evening before, after the puncture, of headache and nausea, but has had a good night for the first time for several months; feels easier. Objective signs the same; 10 c.c. spinal fluid removed; same appearance as before, contains a lot of yellowish pigment granules, moderate lymphocytosis with a few polymorphs. Feb. 21st, says she was much better after last puncture; paraplegia still in *statu quo*. Lumbar puncture: 8 c.c. of paler fluid withdrawn; cellular elements as before, no free pigment granules nor fibrin.

Feb. 23rd, patient can move foot and leg; muscular spasms are seldomer, and less intense; anaesthesia has disappeared. March 7th, progress continued; 8 c.c. spinal fluid withdrawn: clear, fewer lymphocytes, no polymorphs. March 8th, great improvement since the day before; has had no spasms during the day, voluntary movements easier; reflexes in *statu quo*. Mercurial treatment stopped. March 18th, 8 c.c. spinal fluid withdrawn: no small lymphocytes, few large lymphocytes. May 4th, 10 c.c. spinal fluid withdrawn, after which patient says she improved greatly. June 15th, reflexes in *statu quo*; can walk with two sticks. Oct. 16th, has been periodically having mercurial treatment; walks without stick; sphincters normal; tendinous reflexes active; no spinal epilepsy; Babinski's sign present on both sides; abdominal reflexes lost; pupils in *statu quo*. 8 c.c. spinal fluid withdrawn: slight lymphocytosis with relatively numerous large lymphocytes.

Discussing the case, Babinski holds that the spinal fluid demonstrated the presence of a lymphocytic fibrinous meningitis of hæmorrhagic nature as shown by the greenish colour. The lesion must be in the dorsal and dorso-lumbar region, and might be either a meningitis giving rise to a fibrinous exudate compressing the



cord, or a meningo-myelitis. The very intensity of the symptoms showed that the nervous elements were rather irritated than destroyed. In view of the pupil symptoms, the syphilitic nature of the infection is probable, of which chronic meningeal lesions remain. Babinski claims that the withdrawal of the cerebro-spinal fluid had a very important therapeutic action, as was evident from the speedy disappearance of the fibrin: without the puncture one might have diagnosed a lesion situated exclusively in the cord.

C. MACFIE CAMPBELL.

**POST-DIPHTHERITIC CHRONIC BULBAR PARALYSIS.** WILFRED (190) HARRIS, *Brain*, Winter Number, Part civ., 1903.

AFTER a short review of some published cases of chronic bulbar paralysis in which the upper facial muscles were involved, two cases are described in young women, in which a chronic bulbar paralysis developed after an attack of ulcerated sore throat, the illness in one of the cases being recognised by the medical attendant as diphtheria. In Case 1, the symptoms commenced six weeks after an attack of ulcerated sore throat, with nasal regurgitation, when the soft palate was found to be paralysed. Gradually afterwards the lips, eyelids and tongue became paralysed, and also the pharyngeal muscles. There was never any weakness of the limbs, or ocular palsy, or ptosis, and she has been in *statu quo* for the last four years, reaction of degeneration being present in the lips and eyelids. In Case 2, the symptoms commenced three weeks after an attack of diphtheria with paralysis of the soft palate, followed by clumsiness of the tongue and paralysis of the eyelids and lips, with escape of the middle facial muscles, exactly as in Case 1. After twelve months she improved slightly, the faradic reactions in the paralysed muscles being only diminished, but she remained unable to purse up the lips or to close the eyes for six years, the symptoms rapidly clearing up after the birth of her first child, a year after marriage. She has remained quite well since, the power of the muscles of the face being now quite normal. The differential diagnosis of these cases from myasthenia gravis is discussed, the absence of ptosis, or of weakness of the jaw or limbs, with the non-variability of the symptoms and the absence of the myasthenic reaction in Case 1, and the presence of the reaction of degeneration, distinguishing these cases from myasthenia, and suggesting a form of nuclear lesion, not sufficiently severe in Case 2 to prevent recovery after a long interval, under the altered nutritional conditions of pregnancy. The diagnosis of diphtheria in the two cases rests upon inference. The sore throat was recognised as diphtheria in one of the cases, but the commencement

of the symptoms with palatal palsy a few weeks after the sore throat seems to make the diagnosis of diphtheria most probable.

Another point discussed at length in the paper is the nuclear supply of the upper facial muscles. Mendel's hypothesis that the upper facial muscles are supplied, not from the facial nucleus, but from the posterior portion of the third nucleus, is shown to rest on insufficient evidence, modern experimental work undertaken by Nissl's method, as well as recent post-mortem results, failing to show any connection between the upper facial muscles and the third nucleus, but proving their origin to be from the lower cell group of the facial nucleus.

AUTHOR'S ABSTRACT.

**TWO TUMOURS OF THE BRAIN.** T. M. T. M'KENNAN, *Journ. of* (191) *Nerv. and Ment. Dis.*, May 1904, p. 301.

THE author describes a case in which a cyst of the right occipital lobe, "probably hæmorrhagic in origin," was successfully treated by operation; also a case of endothelioma of the left frontal lobe which was removed, the patient dying, however, four days later. He has collected 24 cases of occipital tumour reported since 1893; 11 of these cases were operated upon. Death occurred in 17, recovery in 3, while in 4 the result is not stated.

EDWIN BRAMWELL.

**DISSEMINATED SCLEROSIS WITH SPECIAL REFERENCE TO** (192) **THE FREQUENCY AND ETIOLOGY OF THE DISEASE.** BYROM BRAMWELL, *Clinical Studies*, April 1, 1904, p. 193.

THIS paper contains remarks on the Relative Frequency of Disseminated Sclerosis (vide *Rev. of Neurol. & Psychiat.*, 1903, p. 12) and upon the etiology of the disease. The author's conclusions are based upon 110 cases of disseminated sclerosis which have come under his own personal observation.

In this series of cases, 67 were females and 43 males. With regard to age, in 47, or 42 per cent., the disease commenced between the ages of 21 and 25 years (inclusive); in 76, or 69 per cent., between the ages of 16 and 30 years (inclusive); and in 85, or 77 per cent., between the ages of 16 and 35 years inclusive. In 3 cases the first symptoms appear to have developed at the ages of 2, 7 and 11 years respectively. In only 4 of the 110 cases did the disease develop (or appear to develop) after the age of 50.

A study of the age at which the symptoms were first noticed

in relation to the sex of the patient shows: (1) That in those cases in which the disease commenced before the age of 16 there were more males than females—5 males and 2 females—(the numbers are so small that the relative frequency may have been a mere coincidence). (2) That in those cases in which the disease commenced between the ages of 16 and 30 (inclusive), there were many more females than males—24 males and 52 females. (3) That in those cases in which the disease commenced after the age of 30 the number of males and females was practically the same—14 males and 13 females.

With regard to marriage, the author finds that of 102 cases, in 76 the patients were unmarried at the time when the symptoms were first noticed.

As to heredity, he remarks: "In none of my cases did the disease appear to be directly inherited, and in comparatively few cases did the patients come of a nervous stock; in the majority of the cases the patients were, prior to the development of the symptoms, non-nervous, healthy young men and women."

An enquiry into occupation failed to reveal any special influence on the production of the disease. "The great majority of the patients were either comfortably or fairly well circumstanced as regards their home surroundings." The author's experience coincides with that of almost all observers, viz., that syphilis is very rarely, if ever, the cause of the disease.

An analysis of the more frequent of the "different conditions which appeared to be, or were supposed to be, the cause" in the series of 110 cases may be summarised as follows:—

There was no apparent or alleged cause in . . . . .	39 cases
Febrile and infectious diseases, either alone or with other cause, . . . . .	20 "
Mental worry, grief, mental shock, fright, either alone or with other cause, . . . . .	19 "
Chill, getting wet, either alone or with other cause, . . . . .	14 "
Injury, . . . . .	11 "
Fatigue or physical strain, . . . . .	7 "
Alcoholic excess, . . . . .	2 "
Sexual excess, alone, or with other cause, . . . . .	2 "

It is interesting to note that influenza appeared to be, or was alleged to be, the cause in 9 cases; while in 4 additional cases influenza was associated with an additional apparent or alleged cause. In only 1 case was a history of metallic (lead) poisoning elicited.

The pathology of disseminated sclerosis is briefly discussed, the author inclining to the view that the disease is due to some developmental or congenital defect of the neuroglial or nervous

tissue, which renders it more liable to be affected by irritation than the neuroglial or nervous tissue of the normal individual.

EDWIN BRAMWELL.

**PARALYSIS AGITANS: SOME CLINICAL OBSERVATIONS**  
(193) **BASED ON THE STUDY OF 219 CASES SEEN AT THE**  
**CLINIC OF PROF. M. ALLEN STARR.** T. S. HART, *Journal*  
*of Nerv. and Ment. Dis.*, March 1904.

THIS paper records observations made on a very large number of cases—219. The fact that 219 cases of paralysis agitans have been under treatment in the neurological clinic of one American physician (Prof. Allen Starr), appears to the writer of this abstract, to indicate that the disease is probably more common in the neighbourhood of New York than in England.<sup>1</sup>

As regards antecedents and causal factors, Hart concludes, from the analysis of his cases, that occupation has no bearing on the etiology. In 40 of the 219 cases the disease was attributed by the patient to emotional disturbances (anxiety, worry and fright); in 31 it was attributed to injury. In 87 cases no evidence of any exciting cause could be obtained. From the records of his cases, Hart thinks that emotional disturbances and injury must be regarded as important factors in the etiology of the disease.

Statistics are given regarding the symptoms. Tremor was present in 203 out of 207 cases, in 4 it was absent. The onset of tremor was seven times as frequent in the upper extremities as in the lower. Rigidity was present in 142 cases; in 138 of these tremor was also present, in 4 it was absent.

There was a tendency to fall in various directions in 105 out of 173 cases; in 68 this tendency was absent. The conditions noted were as follows:—Propulsion in 71; retropulsion in 11; lateropulsion in 6.

Pain, when present, was not severe as a rule. It was described as aching or dull by the patient.

Paræsthesia was present in 120 cases. These abnormal sensations were, "pricking, numbness, tingling, flushing, heat and cold." The most common subjective sensory disturbance was the feeling of heat or cold.

In 85 cases "restlessness" was a marked feature.

<sup>1</sup> At the Manchester Royal Infirmary, in a period of ten years, there were only 6 cases of paralysis agitans amongst 18,929 medical in-patients (of these 2430 were cases of disease of the nervous system). Hence the proportion was 1 amongst 2321 medical in-patients, and 1 amongst 405 cases of disease of the nervous system. The total number of deaths from paralysis agitans in England and Wales, during the year 1898, was 341, or 11 per million living.—R. T. W.

As regards treatment, though no cure was obtained, Hart states that "in a number of instances the disease came to a standstill, and remained so a number of years, while many times it seemed evident that the treatment employed materially modified the symptoms, such as tremor and insomnia, rendering the patient's life far more comfortable and happy." Moderate exercise was often of considerable value. Of far greater value was systematic massage, passive movements and hydrotherapy. No improvement was obtained by various forms of electrical treatment.

The drugs which proved of most value were hydrobromate of hyoscine and sulphate of duboisine. These, "for a time at least, diminished the tremor and relieved insomnia."

R. T. WILLIAMSON.

**PARKINSON'S DISEASE (PARALYSIS AGITANS).** Prof. F. (194) RAYMOND, *Nouvelle Iconographie de la Salpêtrière*, No. 1, 1904.

THIS article is the report of a clinical lecture on four cases of paralysis agitans, with an account of the more important views as to the pathology of the disease, and remarks on its treatment.

The first case was that of a female, aged 60, whose occupation had been a polisher of metal, and this had caused overstrain of the right hand only; but the tremor had commenced in the left arm. The second case was that of a man, aged 69. The tremor had commenced in the left hand after great mental depression caused by the loss of a son. The third case was that of a man aged 53; the symptoms had developed after a period of mental anxiety and worry.

Graphic descriptions of the symptoms of these cases are given. Excellent photographs illustrate the article, showing, in the first case, the marked cyphosis; in the second case, cyphosis of a moderate degree, with a stooping attitude; in the third case, the fixed attitude and fixed facial expression. The fourth case presented rigidity of the trunk, with slight lordosis, and was shown as a contrast to the other three cases.

The more recent results of the pathological examination of the nervous system in this affection are considered, and objections to the various theories as to the pathogenesis of the disease put forward. As Raymond does not consider that the pathology of the disease has been satisfactorily explained, an account of these theories does not appear necessary in this abstract. The progress of the disease is described, and the grave prognosis is pointed out; attention is drawn to the long duration of the affection, and to the fatal termination from cachexia, often associated with septic infection from a bed sore or some intercurrent disease, such as tuberculosis. At the present time there is no curative treatment.

The treatment can only be symptomatic. Hyoscyamine, and especially hyoscine, have given temporary relief in a certain number of the cases treated by Raymond. Hyoscyamine may be given in granules containing a milligramme of the drug. For the administration of hyoscine, Raymond refers to the prescription recommended by the abstractor<sup>1</sup>: Hyorcline hydrobromate,  $\frac{1}{2}$  grain, in 6 ounces of chloroform water; dose, two teaspoonfuls, increasing up to three, four, or five teaspoonfuls twice a day, if the drug is well tolerated.

In place of hyoscine, Raymond thinks that hydrobromate of scopolamine is better tolerated by some patients, and is sometimes more efficacious.

For the muscular rigidity Raymond has employed, with a certain amount of success, a treatment consisting of re-education of the muscles, associated with electric baths (static) and currents of high frequency. Arsenic may be of service by improving the general condition. The patient should be placed in good hygienic conditions, physical and moral, and should be spared from all physical and intellectual overstrain.

R. T. WILLIAMSON.

**ACROMEGALY: A CRITICAL RESUMÉ.** G. MODENA, *Riv. (195) sperimentale di Freniat.*, Vol. xxix. Fasc. 3 and 4, 1903.

THIS is an elaborate resumé of the subject of acromegaly. The paper is divided into three parts, of which the first discusses the symptoms, pathological anatomy and clinical course of the disease. These are too well known to require recapitulation here.

The second part deals with the associations and connections between acromegaly and other diseases, including giantism. Amongst the most important diseases associated with acromegaly, either preceding or accompanying it, are myxœdema and exophthalmic goitre. The precise significance of this coincidence is obscure, likewise the connection between the function of the thyroid gland and that of the pituitary body. Diabetes is not an infrequent accompaniment of acromegaly. Various theories have been suggested to account for this, amongst which may be mentioned one ascribing the glycosuria to a lesion of the fourth ventricle, another attributing it to a lesion of the *tuber cinereum*, and a third suggesting a pancreatic origin.

The connection between acromegaly and giantism is one of great interest. The writer of the article inclines to the view of Meige, who pointed out how closely allied the two conditions are, how acromegaly never precedes giantism but, on the contrary, in about 50 per cent. of cases of giantism, acromegaly follows. In

<sup>1</sup> "Paralysis Agitans," Manchester, 1901, p. 45.

both conditions there is, according to Meige, an excessive osteogenetic activity. If the bones are capable of growth in length, the result is gigantism; but if the epiphyses have united and only hypertrophy of the ends of the bones is possible, the result is acromegaly.

The third and last part of the paper deals with the etiology and pathogenesis of acromegaly. It is not uncommon to find a family history of acromegaly, gigantism, nervous or mental disease. Female patients outnumber males in the proportion of five to four. The age of onset is generally after twenty years and before forty, but the latter age is by no means the limit. Infective fevers, syphilis, trauma, exposure to cold, etc., have all been blamed as supposed exciting causes, and the disease certainly seems more frequent in the poorer, ill-nourished classes.

Various theories proposing to account for the disease are then discussed in detail. Klebs's theory, attributing the disease to excessive development of the vascular system, a supposed effect of special endothelial cells in the thymus, suggested to be swept off as emboli to the bones of the extremities and there acting as angioblasts to form new vessels, is untenable. Another theory is that of Freund, according to which acromegaly is a developmental anomaly, coming on during the second dentition. At puberty, the sexual function is supposed to be precocious but of short duration, and thereafter a deviation of genetic activity occurs, resulting in acromegaly, in which disease the long arms, prognathous jaw, anthropoid facies, etc., indicate a reversion to a primitive type. But the age of onset does not necessarily coincide with puberty and this theory therefore falls to the ground. Majendie, Recklinghausen, Dreschfeld and others ascribed acromegaly to a nervous dystrophy, the bony lesions being analogous to those of tabes, syringomyelia and other organic nervous diseases. But, as Tamburini points out, even although the nervous system may play a part in the disease, this does not explain its pathogenesis.

Pierre Marie was the first to point out the constancy of the connection between acromegaly and lesions of the pituitary body, and he came to the conclusion that it is the accumulation of some toxic substance in the nervous system, no longer neutralised by the pituitary secretion, which produces a continuous irritation in the extremities resulting in hyperplasia of bone and other connective tissues. He considered it a disease comparable to myxœdema and deficient thyroid secretion. Tamburini has suggested an elaboration of the preceding theory, to which the writer of the article is inclined to adhere. According to Tamburini's hypothesis, there are in acromegaly two distinct phases. The first is a phase of hypertrophy and functional hyperactivity of the gland, whereby abnormal substances are produced, whose action is to cause growth of bony tissues. This is followed by a later phase

in which profound secondary changes, cystic degeneration, adenoma, etc., occur in the gland, which now ceases to functionate. This is the period when the progressive bony overgrowth stops and cachexia supervenes, inducing the fatal termination. Marie's theory assumes an abolition of function, Tamburini's an excess of function, of the pituitary gland. Modena rejects Marie's hypothesis, pointing out that if abolition of pituitary function were the cause of acromegaly, this latter disease ought to supervene in all cases where the gland is destroyed. But numerous cases are recorded of malignant tumours, tuberculous disease, echinococcus cyst, etc., destroying the pituitary body without producing any bony enlargement or increase of connective tissues. Nor does experimental destruction of the gland cause symptoms of acromegaly. The commonest form of tumour in acromegaly is an adenoma, and in some cases the gland is found merely hypertrophied. Benda states that many cases described as adenomata are really a hyperplasia of one of the three types of cells (the chromophile type) which are normally found in the gland, to which primitive hypertrophy there may be subsequently superadded adenoma, cystic growth or malignant disease. Opponents of Tamburini's theory point out that a small number of cases have been recorded in which the pituitary body was either normal or atrophied. Whether certain of these cases can be explained as instances of moderate enlargement followed by sclerosis, is questionable. Babinski records a case of infantilism with signs of pituitary growth and suggests that this gland has some connection with the sexual function. This may well be so, for in acromegaly there is always some disturbance of sexual activity; amenorrhoea is an early symptom in female cases, whilst in males diminution of sexual power is invariable and impotence not uncommon.

Additional facts, clinical and anatomical, are still required to clear up the obscurity of the functions of the pituitary body. Meanwhile, evidence seems to point strongly to the close connection between pituitary enlargement and acromegalic symptoms. Whether hyperfunctioning of the gland is the cause or the result of the disease, is not definitely established.

PURVES STEWART.

**A CASE OF ERYTHROMELALGIA, ILLUSTRATING ITS RELATION TO RAYNAUD'S SYMPTOM-COMPLEX; "DIFFUSION" OF THE PHENOMENA DURING PERIOD OF EXACERBATION.** F. PARKES WEBER, *Brit. Journ. of Dermatol.*, Feb. 1904.

THE patient was a Jewess, aged 36, free from any sign of alcoholic or arsenical neuritis or organic disease of the central nervous



system. She had suffered for some years from sensations of "burning" in the feet during summer and could not put on her boots without pain. While under observation, July to October 1903, there was more or less persistent pain in the feet and a variable amount of livid mottling, especially of the toes and distal part of the metatarsal region of the left foot; there was habitual hyperæmia of the face and tongue. The left foot was the part mainly involved, but there was occasional flushing or lividity of toes of the right foot, and pains were occasionally present there as well as in left foot. During a period of exacerbation (and more or less "generalisation") of the disease, both hands were hyperæmic and inclined to sweat and both feet were markedly affected—red and turgid with blood, painful, hyperæsthetic, hyperalgesic, and moist with sweating. There was no evidence of obstruction in any of the main arteries of the lower extremities, and no history of loss of blood from lungs, bowel, etc. Various methods of treatment, local and general, were tried, but nothing seemed to have much effect: the condition was aggravated by every attempt to get about on her feet.

Weber regards the case as an "*idiopathic*" form of *erythromelalgia* with occasional tendency to spreading or diffusion of phenomena.

As regards pathology, he thinks it is not unlikely that some arterial changes are to be found in all *really chronic* cases of erythromelalgia and Raynaud's phenomena, but he does not believe that such arterial changes are in any sense primary: the early symptoms can be best explained on the supposition of a vasomotor disturbance due to the central nervous system.

Both from clinical observations and from theoretical considerations, there appears to be no hard and fast line between erythromelalgia and Raynaud's phenomena. Clinically intermediate cases have been recorded. In the *chronic* stage of erythromelalgia the livid mottling may be compared to the "local asphyxia" of Raynaud's phenomena and indicates probably that there is local contraction of arterioles, with engorgement of venules. It seems probable that in the *acute* stage this local contraction of the arterioles is temporarily overcome by increased arterial blood-pressure and active flushing of the whole affected part. Just as the milder forms of Raynaud's phenomena merge into the effects produced normally by cold, Weber thinks it possible to regard the milder forms of erythromelalgia as merging into the phenomena produced in some persons by sudden warming of the feet.

A. W. MACKINTOSH.

**A CONTRIBUTION TO THE STUDY OF THE VISCERAL ANÆSTHESIAS IN GENERAL PARALYSIS.** S. SOUKHANOFF,  
(197) *Rev. Neur.*, 30th April 1904.

THE author gives three examples of the kind of anæsthesia which he has been studying. The first patient, a woman of twenty-one, already in an advanced stage of general paralysis, was delivered of her first child after a painless labour: other cases of the same nature have been recorded. The second patient, who had a pronounced gastric ulcer with copious hæmatemesis, felt no pain and made no complaint. The third case was that of a paralytic, both of whose lungs were the site of advanced tuberculous lesions, but who during life had practically no cough nor spit. According to Soukhanoff the dementia is insufficient to explain the phenomenon which is the result of several factors—the psychical alteration, the anæsthetic qualities of toxines due to auto-intoxication, the general alteration in the nervous centres and in the peripheral nerves.

C. MACFIE CAMPBELL.

**THE VALUE OF ASTEREOGNOSIS AS A LOCALISING SYMPTOM**  
(198) **IN CEREBRAL AFFECTIONS.** W. N. BULLARD, *Journ. Nerv. and Ment. Dis.*, April 1904, p. 241.

THE main facts established regarding the occurrence of astereognosis in lesions of various parts of the nervous system are stated thus:—

1. Astereognosis occurs in affections of the cerebral cortex in the motor region, whether these affections be traumatic or due to other causes. Bullard finds in recorded cases little really conclusive proof of the existence of astereognosis in purely cortical lesions; the evidence is almost wholly clinical, or based upon cases in which the cranium was opened, either directly through trauma or by trephining.

2. There is strong evidence clinically that astereognosis occurs in many subcortical lesions, and also in capsular and thalamic lesions. It is a common accompaniment of organic hemiplegia.

3. Astereognosis may occur in lesions of the medulla, as in the case of chondro-sarcoma reported by Dercum.

4. Astereognosis occurs clinically in locomotor ataxia, ataxic paraplegia, and possibly other diseases of the spinal cord. It has been found in multiple neuritis.

Regarding the value of astereognosis as a localising symptom, "we can only say that when it occurs alone it rather suggests subcortical affections, usually pressure. When in combination with other symptoms, it may serve to confirm or strengthen the

diagnosis, but only aids in determining the localisation within the wide limits mentioned."

Notes are given of a case in which astereognosis of the left hand was one of the earliest and most prominent symptoms. The patient was a man aged fifty-eight, who began to notice numbness of the left arm and leg, and occasionally of right leg, and his legs often felt as if asleep. Six weeks later, astereognosis of the left hand was noticed and some loss of muscular sense in the same hand, his gait became unsteady and his power of walking less. When seen by Bullard ten weeks after onset, he was found to have double optic neuritis; the right limbs were normal, and there was no definite weakness of the left limbs, although he dragged the left foot in walking. In the left arm the sensations to touch and pain were normal, the temperature sense good, the "muscle sense" diminished; he could not distinguish between one or more points touched simultaneously over the whole limb; he could not touch the nose with the left forefinger when the eyes were closed; there was astereognosis of the left hand and forearm; in the left leg the sensory conditions were essentially the same, except that the sensation to heat was diminished and the muscular sense was not impaired; the sense of pressure (as indicated by weight) was diminished, the sense of location of touch was lost; he felt a pencil laid on lengthwise as a point (astereognosis); some static inco-ordination, knee-jerks exaggerated, Babinski reflex present.

The course of the case till death, two months later, showed a very distinct improvement in the sensory conditions, and he became able to distinguish objects in his left hand fairly well; left hemianopsia was noted, his left limbs became almost totally paralysed, and he gradually failed.

At the autopsy a gliomatous tumour was found on the right side of the corpus callosum, extending 8 cm. from before backward, beginning at a point 5.5 cm. posterior to the frontal pole, bounded on the inner aspect by the outer wall of the right lateral ventricle, and with ill-defined external margin, which in places approached somewhat closely to the cortex. The structures directly involved in the tumour were: (1) the genu, tapetum and forceps major of the corpus callosum on the right side, and (2) the corona radiata and centrum semiovale for a distance antero-posteriorly corresponding with the extent of the lateral ventricle.

A. W. MACKINTOSH.

**DEAF-MUTISM AND COËN'S IDIOPATHIC ALALIA.** G. (199) PERUSINI, *Riv. di Patolog. Nerv. e Ment.*, 1904, p. 49.

THE patient was a boy of seven years, born naturally at full term, the father being alcoholic. All the other children of the family

were slow in learning to speak; no nervous or mental disease was present in any other relation. At the age of one year, the patient, who had begun to walk alone, had a fall, striking his head on the occipital region, and it was a year after this accident before he again began to walk. At the age of five and a half years he had a series of six convulsive attacks without paralytic sequelæ, but after the first convulsion, from being docile and intelligent, he became passionate and disobedient, sometimes violent. He was never able to articulate even a single word. The only vowel-sound he could form was A; the only consonants, M and T. Beyond these he made inarticulate guttural noises. On examination he was found to be "almost normal" in intelligence. Hearing and all the other special senses were acute. No other abnormality was detected in the nervous system. There was a cardiac bruit, indicating mitral stenosis. An extraordinary degree of dermographism was present.

After quoting Morselli's classification of speech disorders, based on that of Kussmaul, Perusini places his case in the category of alalia—congenital disability to emit articulate sounds. Both deaf-mutism and idiocy could be excluded. Alalia, as originally described by Coën, is a form of mutism occurring between the ages of three and ten years in children of normal physique and intelligence, without deafness, with all the peripheral organs of articulation normal, and without paralyses or atrophies in any part of the trunk or limbs. Apart from hereditary neuropathic tendencies, no definite etiological factors can be laid down. The diagnosis is easy. The mental condition is normal, or, to be more accurate, as near normal as is conceivable in a patient who has never had the mental exercise of articulate speech. The pathological anatomy is as yet unknown.

Perusini then proceeds to discuss the various theories which have been suggested as to the pathogenesis of the disease, but these are not adapted for the limits of a short abstract.

PURVES STEWART.

#### **CONJUGATE DEVIATION OF THE HEAD AND EYES, WITH (200) HOMONYMOUS HEMIANOPSIA: ITS SENSORY ORIGIN.**

H. DUFOUR, *Rev. Neur.*, April 15, 1904.

DUFOUT gives the cases of two patients presenting a right-sided homonymous hemianopsia, with conjugate deviation of eyes and head to the left: the occipital pole on the left side showed deep or cortical lesions, explaining the hemianopsia. In the first case the left-sided deviation was preceded by a right-sided deviation during the irritative stage of the lesion; the fact that the patient

could voluntarily correct the deviation showed that it was not of a paralytic nature but depended on the hemianopsia, the lesion at first irritating the paths of conduction of optic stimuli had reflexly caused a temporary deviation to the right.

In the second case the conjugate deviation of the eyes and rotation of the head disappeared with the hemianopsia; in this case the patient could, on closing the eyes, correct voluntarily the rotation of the head, but not the deviation of the eyes. One cannot, therefore, assume the existence of a single cerebral centre for these two movements.

Dufour agrees with Bard that this conjugate deviation is due to the active contraction of the muscles of the non-affected side, stimulated automatically by the unilateral functioning of the sensory centres due to unilateral loss of central perceptions or of the reflex activity of the sensory motor centres. The fact that hemianopsia often exists without the presence of the abnormal attitude does not invalidate the sensory explanation of the latter: the syndrome is only observed in comatose or semi-comatose cases. Not only must the path of optic conduction be injured, or the cortex itself, it is also necessary that the whole sensory life of the hemisphere be impaired, thus giving the preponderance to the intact reflectivity of the other hemisphere.

C. MACFIE CAMPBELL.

**CONCERNING A PECULIAR REFLEX PHENOMENON IN THE  
(201) EXTREMITIES IN CENTRAL ORGANIC PARALYSIS.** VON  
BECHTEREW, *Neurolog. Centralbl.*, 16th May 1904, p. 434.

SEVERAL years ago von Bechterew described (*Neurolog. Centralbl.*, 1895, No. 24) a symptom which he had observed in the paralysed arm in cases of hemiplegia in which the reflexes were exaggerated without marked contractures.

If the hemiplegic allows his paralysed arm to remain as flaccid as possible, and if the observer flexes the arm at the elbow-joint and then suddenly leaves go, the arm does not immediately drop to the vertical as is the case with the healthy limb. The arm drops so far, but before it reaches full extension the tendon of the biceps suddenly becomes tense and the arm is momentarily arrested before it resumes its downward course; sometimes indeed the contraction of the biceps is sufficient to produce a visible movement at the elbow in the direction of flexion. This sudden upward jerk can be very distinctly felt if the physician places one hand on the patient's shoulder and with the other holds the fingers and suddenly extends the arm.

Mohr (*D. Ztschr. f. Nervenheilk.*, 1901, H. 2-4, S. 204) has de-

scribed a similar phenomenon. If the observer grasps the paralysed arm by the hand and brings it suddenly into the supine position, he receives the impression that supination takes place in two acts. Soon after the commencement of the movement, he experiences a resistance which it is necessary to overcome to complete the movement. Von Bechterew claims that this phenomenon is of the same nature as that which he described in 1895. In that paper he stated that similar phenomena might also be observed in other muscles.

The author agrees with Mohr that these phenomena are of reflex origin and that they do not occur in functional hemiplegia.

EDWIN BRAMWELL.

**IRRITABILITY OF THE FACIAL MUSCLES (FACIAL RESPONSES (202) AND REFLEXES).** CHARLES L. DANA, *N. Y. Med. Journal* July 25, 1903, p. 161.

THE author includes under the term, "Facial Responses," first, the contractions produced by a direct blow upon the muscle or its motor nerve; second, the myotatic response produced by a blow upon a tendon, giving rise to a direct contraction by virtue of the disturbance of the muscle tonus; and third, the simple reflex caused by irritation of a sensory or excito-reflex nerve. The second and third responses are absent when there is any interruption of the sensory-motor reflex arc.

The writer has studied the facial responses upon normal individuals and upon cases of insanity, many types of nervous disorders, also upon cases in which there was a total paralysis of the seventh nerve, and cases in which there was a total paralysis of the fifth nerve. Through his study, particularly upon the last group of cases, the writer comes to the conclusion that all the normal responses obtained by mechanical irritation of the facial nerves and muscles except one, are direct muscular reactions and not reflexes. A diagram is given, showing the location of these various direct muscular responses. The single facial reflex which is uniformly present in adults, is the supra-orbital reflex (M'Carthy's). In some normal individuals there is also a naso-mental reflex; this naso-mental reflex, however, is usually an evidence of a pathological condition, and is seen especially often in paresis and in other psychoses, with excitement, also, in some neuroses, where there is considerable psycho-motor irritability, as in Basedow's disease. A third facial reflex is still more rarely observed, namely, the fronto-mental reflex, a blow on the supra-orbital nerve causing an elevation of the lower lip. Other rare facial reflexes such as the pouting-reflex in infants are described. A list of these is given

and diagrams showing their location are published. The nasomental reflex has some clinical value, as indicating an abnormal excitability of the cortex, or, perhaps, of the subcortical centres.

Some tact and skill are required in eliciting the reflexes properly. The author uses a simple rubber percussion hammer.

AUTHOR'S ABSTRACT.

## PSYCHIATRY.

**THE ETIOLOGY OF SIMPLE IDIOCY COMPARED WITH THAT (203) OF CEREBRAL INFANTILE PARALYSIS.** W. KOENIG, *Allg. Ztschr. f. Psychiat.*, Bd. 61, H. 1 and 2, 1904.

IN previous papers the author has maintained that there is a continuous series of cases extending from cerebral infantile paralysis with normal mental capacity to simple idiocy. If this contention is accurate, the etiological factors in the two groups should be very similar. In order to test this point he has analysed these factors in 260 cases of simple idiocy observed by himself, and compared them with those in 70 cases of cerebral infantile paralysis without idiocy.

As true etiological factors, he understands only those which have been shown to be capable, independently of other influences, of causing either cerebral infantile paralysis or idiocy. All others he regards as predisposing or co-operating factors.

Only three true etiological factors of cerebral infantile paralysis are known, namely, (a) difficult or asphyxial birth, (b) head traumatism, and (c) infections. In regard to these factors, comparison of the two morbid conditions, cerebral infantile paralysis and simple idiocy respectively, brought out the following results: difficult or asphyxial birth, 11·4 per cent. and 10 per cent.; traumatism, 5·7 per cent. and 2·6 per cent.; infections (excluding syphilis), 7·1 per cent. and 3·4 per cent.

A comparison of the occurrence of the predisposing or co-operating causes in the two series showed mental or nervous disorders in the ancestors in 28·5 per cent. and 32 per cent.; hereditary history of phthisis in 14·4 per cent. and 13·8 per cent.; paternal drunkenness in 23 per cent. and 15 per cent.; psychical traumatism sustained by the mother during pregnancy, 23 per cent. and 12·5 per cent.; somatic traumatism sustained by the mother during pregnancy, 2·9 per cent. and 3 per cent.; consanguinity of parents, 1·4 per cent. and 1·1 per cent.; first child in 27·1 per cent. and 17·6 per cent.; premature birth in 10 per cent. and 6·5 per cent.; illegitimate birth in 10 per cent. and 6·5 per cent.; child feeble from birth, 15·7 per cent. and 10 per cent.;

the child a late or the last member of a large family, 10 per cent. and 16.9 per cent. ; mental or nervous disorders in the brothers or sisters, 7.1 per cent. and 30.7 per cent. ; phthisis or scrophulous conditions in the brothers or sisters, 5.7 per cent. and 2.3 per cent. ; death of several other members of the family in infancy, or probable occurrence of abortions, 35.7 per cent. and 16.8 per cent.

In both forms of disease, syphilis may be the sole etiological factor ; it often, however, acts rather as a predisposing cause. In many cases it is impossible to say what degree of importance is to be attached to it. It was ascertained to have occurred in the parents in 7 per cent. of cases of paralysis and in 10.7 per cent. of cases of simple idiocy.

The author recognises that the statistics regarding simple idiocy may to some extent be affected by the circumstance that epileptic fits occurring at an early age tend to cause arrest of mental development.

W. FORD ROBERTSON.

**THE ANATOMICAL FACTS AND CLINICAL VARIETIES OF**  
(204) **TRAUMATIC INSANITY.** ADOLF MEYER, *Amer. Journ. Insan.*,  
Vol. lx, No. 3.

THE author gives the cases of thirty-one patients out of three thousand admissions over a period of six years. There is not a traumatic insanity, but various forms of insanity with peculiar characteristics due to trauma. The ordinary names which one applies to the non-traumatic forms of insanity, *e.g.* mania, melancholia, are inappropriate and inadequate to describe these traumatic forms. The author lays stress on the concurrence of several etiological factors in the majority of his cases, and considers the question whether these patients would have developed the mental disorder without the injury. To this he gives a very reserved answer. On the question of a relation between special types of injury and the form of psychosis, Meyer has found that lesions of the convexity produced epileptiform disorders ; diffuse concussions and basal fractures led to initial delirium, or a paranoiac development or intercurrent episodes of a more or a less epileptiform character.

The immediate result of the cranial injury is often a more or less comatose state which leads on to partial consciousness or actual delirium before the return to full consciousness. Where the mental derangement extends over several weeks we have clinically a case of primary traumatic insanity. In such cases Meyer found absence of manic-depressive symptoms, tendency to dream-states, inconsistency of statement, general haziness : all were subject to impulses and restlessness.



The cases which developed secondary traumatic insanity showed a variety of psychoses, *e.g.* an atypical disorder resembling dementia præcox, epileptiform psychosis, great susceptibility to alcohol, acute hallucinations with hysterical attacks, neurasthenic irritability with melancholic attacks; in several patients traumatism played a part in causing the development of an atypical paranoiac symptom-complex.

The author suggests the following scheme of studying these cases clinically:—(1) Make an inventory of a patient's mental possibilities, analysing his methods of mental working. (2) Study the vaso-motor neurosis of Friedmann: has the case headache, dizziness, flushing, intolerance to alcohol, congestive attacks? (3) Examine for the explosive diathesis of Kaplan where the patient reacts in an exaggerated way varying from bizarre conduct to a real epileptic fit. (4) Investigate dream-states. (5) Consider in the etiology the types of insanity in the family, the constitutional characteristics of the patient, history of alcoholism and syphilis, the extent of the injury and its immediate after-effects, the influence of litigation. C. MACFIE CAMPBELL.

**HYPOCHONDRIASIS WITH DELUSIONS OF PERSECUTION.** A (205) late form of Paranoid Dementia. LUGARO, *Riv. di Patol.*, Vol. ix. f. 3.

WE have in this paper an enquiry into the condition of hypochondriasis with delusions of persecution, and its relation to paranoid dementia, and the melancholia of the involutive age (Kraepelin).

This condition is seen principally in women about the time of the climacteric, and occasionally in men at the corresponding period of life. It manifests itself by a series of hallucinations, chiefly kinesthetic and visceral, and by a delirium of persecution, which is closely associated with the hallucinations.

These hallucinations may arise through any form of sensation by which we appreciate the condition of our body, and the changes which take place in and around it. Sometimes they are of one kind only; but at other times, all the impressions received seem to be misinterpreted, and the whole personality becomes altered. Pseudo-hallucinations, psycho-motor hallucinations are occasionally met with.

The delusions always have a persecutory character—persecution by physical means and by poisoning, delusions of possession and of bodily disease.

In these patients the power of attention is deficient, their judgment is much impaired, and their whole conduct is dominated by their hallucinations and delusions. Their affective state is

slightly depressed; they are irritable, and they are troubled with a painful restlessness. Sleep is disturbed at first. Their memory, however, is not impaired, and they are never dirty.

After a time the hallucinations and delusions lessen, and the patients may pass into a condition of indifference. Very similar symptoms are also found in the early stages of paranoia, but the further course of this disease is very different. In paranoia the hallucinations disappear, and their place is taken by pseudo-hallucinations. The patients are no longer subjects of persecution, but have become expansive, and they are now kings, warriors, inventors, etc.

Nothing like this occurs in the hypochondriac. Here the patients may lose their hallucinations and become calm, but they do not alter in character. Moreover, there is not in them the verbal confusion, the disordered ideation, which is so characteristic of paranoia, and they do not show any catatonic symptoms.

There are points of contact also between the hypochondriac and some cases of melancholia of the involutive age, in whom the fundamental note is not an affective depression, but consists of a series of hallucinations and accompanying delusions of persecution.

In considering all these forms of mental disease, the author suggests that the limits of dementia præcox should be enlarged, and he further suggests that the differences between the hebephrenic, catatonic, and paranoid conditions must be attributed to difference of reaction in brains of different ages to the same morbid agent.

The most precocious forms are the hebephrenic, then come the catatonic. The paranoid cases are the next, at about the age of thirty, and the class of cases dealt with in this paper appears still later in life. He also states that the earlier in life the disease shows itself, the more rapidly does dementia supervene, and the more marked does it become.

R. G. Rows.

**SENILE DEMENTIA: A CLINICAL STUDY OF TWO HUNDRED (206) CASES WITH PARTICULAR REGARD TO TYPES OF THE DISEASE.** WILLIAM PICKETT, *Journ. Nerv. and Ment. Dis.*, Vol. xxxi. No. 2, Feb. 1904.

Two hundred cases of senile dementia are carefully analysed by means of tables, and separated from other acute and chronic psychoses of old age. After a description of the simple or pure form, attention is called to the various types in their order—(1) simple-confusional, (2) excited, (3) depressed, (4) paranoid—which order is gained by consideration of a table of special

symptoms met with in these several types. The author had great difficulty in separating simple from confusional cases; so combining the two produced the "simple-confusional type," a group which contained 143 of the 200 cases. He finds this graded series to be a natural one, "for the records of knee-jerks range the four types of the disease in this same series, while the family histories of insanity expressed in percentage support it strictly." From the results of his observations the following laws are deduced: (1) physical deterioration in senile dementia is greatest in the simple-confusional type, and progressively less in the excited, depressed and paranoid types; (2) hereditary degeneration is least in the simple-confusional, and progressively greater for the others in the same order—excited, depressed, paranoid. He does not find from his statistics that the simple-confusional cases are "oldest in years," and the paranoid youngest. Special attention is called to the paranoid type, as it is so little dwelt upon by the standard authors, and two cases are quoted at length, in which the delusions caused to be disagreeable or dangerous to their families, patients who in an asylum would be regarded as "uninteresting" senile dementes.

C. H. G. GOSTWYCK.

#### **THE IMPORTANCE OF LUMBAR PUNCTURE FOR PSYCHIATRY.**

(207) PROF. NISSL (Heidelberg), *Centralbl. für Nervenheilk. u. Psych.*, April 1904.

NISSL gives the results of his examination of the cerebro-spinal fluid of patients in the Heidelberg Psychiatric Clinic. One hundred and sixty-six cases were tapped, 218 punctures being made. Omitting cases where there was some mishap in the technique, 128 cases are tabulated, the number of punctures being 163. Attention was paid to the pressure of the fluid, the presence or absence of cells in it, and the albumin present. No special instrument was used to measure the pressure, and without that Nissl holds it impossible to do more than estimate extreme variations of pressure. Criticising Schaefer's results gained with Quincke's method, he is of opinion that the measure of the exact pressure is little help in diagnosis.

To test for albumin in the first 58 punctures, a saturated solution of magnesium sulphate was added to the fluid, which was then filtered and boiled. In all these cases the fluid remained quite clear on the addition of the magnesium sulphate. In the 17 cases of general paralysis examined, boiling invariably produced an opalescence or turbidity. The cases of dementia præcox, delirium tremens, hysteria, imbecility, gave negative results with this test for the presence of serum albumin. The cases of meningitis and two syphilitics gave a positive result, as did also a case of arterio-

sclerotic dementia with a negative cytological result. The other cases, with negative chemical result, gave also negative cytological result. Nissl later employed a saturated solution of ammonium sulphate, a much more delicate test; with this one always gets a positive result, even with normal individuals, showing that even in the normal fluid there is a substance which behaves like albumin. On the contrary, out of 158 examinations, only four cases, and these paralytics, contained serum globulin, shown by the opalescence on the addition of ammonium sulphate in the cold. In all the other cases, general paralytics included, the fluid remained clear on the addition of the sulphate.

Nissl concludes that globulin has no importance in normal or pathological cerebro-spinal fluid.

To estimate the total quantity of albumin present, Nissl centrifuged the fluid in a finely drawn-out tube, after the addition of Esbach's reagent.

Out of 68 punctures, increased albumin was found in all the cases of general paralysis (14), one catatonic, two senile dement, one epileptic, one arterio-sclerotic dement, and a case of brain syphilis. The chemical and cytological results are not always parallel; but increased albumin, with negative cytological examination, is more common than to find no albumin, with an increase in the cellular contents. In three cases of senile dementia in arterio-sclerotic patients, and in one arterio-sclerotic dement with no cells present, the albumin was much increased. Nissl formulates the question whether the albumin is increased in the purely hyperplastic form of meningitis with no cellular exudate.

Before giving the results of his cytological examination, Nissl criticises the technique employed, showing its various fallacies: he insists that the cells are of necessity badly fixed, and cannot agree with E. Meyer in his description of the elements seen. He also holds Ravaut's "réaction discrète" too absolute and as not entitling to a definite opinion: if four to six lymphocytes are seen in the field under the immersion, *non liquet* is all we can say.

Nissl's results agree with those of the majority of observers. In sixty punctures made on twenty-eight paralytics the result was only once negative: in that case a second puncture made twenty days later gave a positive result. The number of elements was found to vary from time to time in the same patients with no obvious explanation. A single negative result, then, does not prove the absence of cells: if there be a suspicion of general paralysis and the first puncture be negative, it should be repeated more than once. As in slowly progressing cases of paralysis the amount of the cellular infiltrate in the adventitial sheath of the vessels diminishes, so it is quite intelligible that at the same time the number of elements in the cerebro-spinal fluid should diminish.

Frequent examination of the fluid in well-marked cases is to be recommended. In one case of general paralysis there were numerous polymorphs present: previously only lymphocytes had been present and subsequently the polymorphs disappeared, the change in the fluid being accompanied by a change in his clinical condition. Does the cerebro-spinal fluid give us any help in diagnosing between certain cases of chronic alcoholism and general paralysis? Nissl comes to no conclusion, as his material was insufficient: but reviewing the literature, he finds two undoubted cases of chronic alcoholism with positive cytological result. He then raises the question as to the value of the examination of the cerebro-spinal fluid in the diagnosis between brain syphilis and general paralysis, but leaves it open.

After analysing twenty-three difficult cases of doubtful diagnosis, Nissl concludes that the examination of the fluid is a valuable aid to the diagnosis of general paralysis, but that its general diagnostic importance is moderate. If psychiatry is to gain much from this new method, the direction of investigation must change. We must first discover the relation of the increase of the cellular elements to the pathological process, and the first step is to find the source of these elements. According to the French authors, the presence of cellular elements demonstrates a meningeal irritation: polymorphs show that this is an acute and intense irritation. The increase of the cellular elements in the cerebro-spinal fluid in a meningitis where we have cellular exudates, seems to be the effect of the latter. Nissl insists on more precision: we know nothing of the production of the cerebro-spinal fluid, the arrangements of the roof of the fourth ventricle are still disputed: in normal adults one finds not only mononuclear lymphocytes, but also "epitheloid cells." What is their origin? Further study is indicated of the histo-pathological changes in the meninges, the choroid plexus, etc.: it is insufficient to determine the existence of a cellular infiltrate; the elements must be analysed, their origin, progressive and regressive changes and their mutual relations must be established.

The cells which are found in the cerebro-spinal fluid may not have an identical origin and may vary according to their source. The technique must be improved, so that the elements can be better studied. Nissl and Devaux have managed to demonstrate the elements sufficiently for the purpose. It is too arbitrary to say that the cellular contents must come from meningeal irritation: in tabes the posterior meninges are thickened, but this is, in many cases, a purely hyperplastic process with no cellular exudate and no inflammatory condition of the roots: how to account for the cells in the cerebro-spinal fluid? Nissl cites a case with positive cytological result in which the membranes on microscopical ex-

amination showed no cellular changes. In insular sclerosis a positive result is common without there being a question of the membranes being touched. In syphilitics, with nothing abnormal to be made out on clinical examination, a positive cytological result does not entitle us to say that the meninges are touched. There are cases of chronic alcoholism very like general paralysis in which the cytological result is positive: yet Nissl has never found inflammatory processes with a cellular exudate on microscopical examination.

Further study of the elements present may in future help us in diagnosing between the inflammatory and non-inflammatory forms of brain syphilis: the latter from general paralysis. All that we can say just now is, that a negative or doubtful cellular examination is more in favour of the non-inflammatory forms of brain syphilis than of general paralysis. Nissl, on the basis of his histopathological work, suggests that the inflammatory forms of brain syphilis would present the same cytological and chemical characters as the majority of paralytics. He calls attention to the difficulty of understanding the occasional presence in the cerebrospinal fluid of general paralytics of numerous polymorphs, while a cellular exudate of such cells is never found in general paralysis unless complicated with a septic process: there was no suspicion of such in Nissl's own case.

After suggesting these further lines of investigation, Nissl considers the practical point whether in an asylum the doctor should perform lumbar puncture as an ordinary mode of examination, or should first ask permission of the patient or relatives. Definite data as to the result of the puncture were got from seven normal individuals, chiefly doctors whom Nissl punctured. One doctor felt no inconvenience from it, although he did not lie down after it. The others, after an interval of several hours, were troubled with headache, nausea and vomiting. In one case the symptoms lasted more than a week. Analysing the mishaps collected by Maystre in his Thesis, said to have been caused by lumbar puncture, Nissl concludes that no permanent damage has been shown to be due to the puncture; but as its effects may for a time be disagreeable, the permission of the patient's friends should always be got.

C. MACFIE CAMPBELL.

### TREATMENT.

**THE SERO-THERAPY OF EPILEPSY BY CENI'S METHOD.**  
(208) MAZZEI, *Riforma Med.*, No. 16, 1904.

BEFORE giving an account of his own work the author briefly synopsis Ceni's views, and then gives the results obtained by other observers who have used the same method.

Mazzei's cases were all well established ones on whom every variety of medical treatment had been tried. Their own serum was used for injection, except on a few occasions when that of another epileptic was employed. No change was made in their mode of living or diet, and if bromide was being taken its use was continued in exactly the same doses.

Of the five cases treated and studied, four were greatly improved. The number and intensity of the fits decreased, the general health improved, and the body weight increased.

Mazzei agrees with Ceni in considering that a permanent cure is not to be expected in the great majority of epileptics, especially in adults and in those with marked signs of degeneracy. He thinks, however, that Ceni's method is a useful adjunct to other forms of treatment when these are insufficient. It enables many patients to follow their occupation and live at home.

DAVID ORR.

**ON THE EXPERIMENTAL USE OF THE ANTI-SERUMS IN  
(209) ACUTE INSANITY.** LEWIS C. BRUCE, *Journ. Ment. Sci.*,  
April 1904.

THE author used the anti-streptococcus serum, anti-staphylococcus serum, and the anti-bacillus coli serum. The subcutaneous injection of anti-bodies in acute insanity was of no value, even in cases where a definite agglutinine was discovered in the blood of the patient and the appropriate anti-body injected.

Anti-streptococcus serum was administered orally to five patients suffering from fully developed acute mania in doses ranging from 10 to 20 c.c. without any benefit; and, in any case of acute mental disease where the symptoms are severe, serum treatment is of no value. In two cases, however, which threatened to relapse, 10 c.c. doses of anti-streptococcus serum reduced the pulse ten to twelve beats per minute, lowered the temperature a degree, and apparently cut short the attack. Two further cases of mania which had recovered to a certain point, but showed every evening a tendency to loss of self-control with a quick pulse and slight rise of temperature, were treated with 10 c.c. doses of anti-streptococcus serum given at 4.30 P.M. to anticipate the rise of pulse and temperature. Both patients benefited by the treatment; their pulses did not show the evening rise, there was less restlessness, and both made rapid recoveries. The action of the serum in these cases was not specific, as shown by the fact that anti-bacillus-coli and anti-staphylococcus serums were administered with the same result.

Three cases of katatonia were treated with large doses of a

serum obtained from a goat immunised to a coccus isolated from the blood of a case of katatonia. The serum produced a marked fall in temperature, but no mental improvement was observed.

These anti-bodies, when administered by the mouth, have a distinct hypnotic action.

H. DE M. ALEXANDER.

**CLINICAL STUDIES ON THE THERAPEUTIC ACTION OF  
(210) CURRENTS OF HIGH FREQUENCY AND HIGH TENSION  
"DANS LES MALADIES PAR RELENTISSEMENT DE LA  
NUTRITION." E. BONNEFOY (de Cannes), *Annales d'Electro-  
biologie et de Radiologie*, 1903, No. 5, p. 513.**

AN introduction which contains nothing of importance, but which gives the author an opportunity of expressing his views on cellular metabolism, is followed by a superficial sketch of the physiological action and therapeutic effects of high frequency currents. Thereafter some eight-and-twenty cases of arthritism are recounted, in which the writer was able to relieve or cure the patient by this form of electrical treatment after many other therapeutic measures had been tried, in some cases for years, with little or no benefit. The name "Arthritism" is defined as "a diathesis or morbid temperament which reveals itself in a great variety of affections, all of which, however, are occasioned by functional derangements of the cell characterised by excessive absorption and insufficient disassimilation," and it includes within its comprehensive bounds, gout, gouty rheumatism, biliary and urinary lithiasis, obesity and diabetes as its principal maladies, and as subsidiary ones the various forms of chronic rheumatism, asthma, emphysema, some circulatory disorders, certain nervous diseases such as migraine, neuralgia and neuritis, several gastric troubles, and a variety of skin diseases, *e.g.* urticaria, eczema, psoriasis and alopecia.

The cases specially referred to by the writer of this paper may be classified as follows:—Arthritism with arterial hypertension, 4 cases, all cured; gouty rheumatism and arthritis, 10 cases, 7 cured, 3 improved; gout, gravel, etc., 7 cases, 5 cured, 2 improved; skin diseases, 4 cases, 1 cured; obesity (gouty), 1 case, improved; general arthritism, varied symptoms, 1 case, cured; biliary lithiasis (gall-stones), 1 case, cured.

It thus appears that the treatment has been conspicuously successful in the author's hands, in the class of cases reported. The form of high frequency mainly employed by him was the condensing couch.

In concluding, the writer lays stress on several details regarding the treatment, and these may be summarised under two heads. Firstly, to get the best results one must employ powerful apparatus,



the applications should be made daily, and each should last about fifteen minutes. As recovery progresses the intervals between the applications may be cautiously extended. Under these conditions the effect is in general sedative and not exciting. Secondly, to get these results most quickly one must study the environment of the patient. He should be removed from his ordinary surroundings and placed where he will be free from worry and subjected to the favourable influences of a mild, equable and sunny climate like that of the Riviera, and especially of Cannes.

The obvious inference is, send the patient to a physician in some good health resort as Cannes, who has sufficient experience and good appliances: don't waste time by trying to cure him yourself.

HARRY RAINY.

**ALBANY HOSPITAL, SECOND ANNUAL REPORT OF PAVILION**  
(211) **F, DEPARTMENT FOR MENTAL DISEASES, FOR THE**  
**YEAR ENDING FEBRUARY 29, 1904.** J. MONTGOMERY  
MOSHER, *Albany Med. Annals*, May 1904.

THOSE in this country who are interested in the question of the desirability of making provision for early cases of insanity in our general hospitals, will find much that may well be pondered in Dr Mosher's second report upon the results of the pioneer effort that is being made at the Albany Hospital to establish something intermediate between home and an asylum in which to treat certain cases of mental disease. The high expectations that were expressed in the first report have been fully justified by the results of the second year's work. Great interest in the experiment has been aroused throughout the United States, and its success is already very widely recognised. Dr Mosher is able to state that "from the general expression of approval we have not found one dissenting voice. The agitation for the provision for mental cases in general hospitals is becoming active and widespread. . . . Rarely does a new enterprise command such immediate and unrestricted commendation. We have builded better than we knew, and from the universal approval may take fresh inspiration for continuance and improvement of our work."

The report states that 171 patients were under treatment during the year. They included cases of acute delirium, confusional insanity, melancholia, mania, acute alcoholic delirium, neurasthenia, general paralysis, idiocy, terminal dementia, etc. Of 157 patients discharged during the year, 51 were cured and 43 had improved. Sixty per cent. of the cases were distinctly benefited. 41 patients were discharged unimproved and 19 died.

Dr Mosher insists strongly upon the necessity of admitting

into such wards only patients who desire to be treated therein. "The hospital has not only not sought, but has declined any licence or charter by which it would be placed upon the footing of an institution for the insane. To accept such prerogative would defeat the fundamental purpose." "All patients treated in Pavilion F, except those under commitment awaiting transfer, are admitted upon their own volition, and are entitled to leave the hospital upon their request." "That the Pavilion can be satisfactorily administered upon this voluntary basis is amply attested by the statistical table of results." W. FORD ROBERTSON.

**CRANIECTOMY BY MEANS OF GIGLI'S SAW.** *Arch. Gén. de (212) Méd.*, April 26, 1904, p. 1025.

PROFESSOR G. MARION writes an article in the April number of the *Archives Générales de Médecine* on the different methods of osteoplastic craniectomy; or, as it may perhaps be more correctly termed, craniotomy.

After mentioning the various methods used by certain Continental surgeons, M. Marion sums up enthusiastically in favour of the operation by means of Gigli's saw. In this conclusion we cordially agree, after having tried the various forms of saw, chisel, dental engine, etc., which have from time to time been recommended for the purpose.

M. Marion uses a Doyens perforator to make his preliminary openings in the skull, instead of using an ordinary trephine of small size. In other respects his method is exactly similar to that used by the writer for many years past with one notable exception, and that is in regard to the division of the pedicle of the bony flap. M. Marion snaps this through by force, in some cases sawing through the inner table with Gigli's saw before doing so. The writer has found this method inferior to that which he recommended some fifteen years ago and still uses, viz., the passing of a director, one inch in width, between the pericranium and the skull from one trephine opening to the other at the neck of the flap. This enables the saw to be applied to the neck of the bony flap in the same way as to the rest of its circumference, without any danger of damage to the vessels contained in the soft parts of the neck of the flap, which are responsible for its nutrition.

The inner table of the skull is brittle and will snap straight across if the outer table be divided. The outer table, however, is not brittle and is liable to split considerably when force is applied to smash it across. This fact renders M. Marion's plan less certain and satisfactory than it would otherwise be.

J. M. COTTERILL.

**REPORT OF TWO CASES OF MENINGEAL TUMOUR TREATED  
(213) BY LIGATURE OF THE VESSELS. A. C. BRUSH, *Journ.  
Nerv. and Ment. Dis.*, May 1904, p. 318.**

CASE 1. Patient began about March 1st, 1902, to suffer from headaches, vertigo and vomiting. From that time up to the date of operation he had convulsive fits affecting the left side of the body. There was loss of power in the left arm and hand. The tumour—a hard, dark red growth imbedded in the pia mater and measuring 1 by 1½ inches—was exposed on May 1st, 1902. All the vessels to the growth were ligatured and the wound closed. During the past year there has been no return of the convulsions, headache, vertigo, or pains; but the loss of power in the left arm has not improved.

Case 2. A patient who for seven years had suffered from slight general clonic spasms with no apparent loss of consciousness. These were preceded by a peculiar feeling in the left leg, and were followed by a temporary flaccid paralysis of the left limb. The attacks had been more severe and more frequent for two years previous to operation. The patient had a partial flaccid left hemiplegia. A cortical tumour was diagnosed and exposed by operation. The growth was similar to that met with in the last case, and on May 28, 1902, all the vessels supplying it were ligatured. The fits have not recurred since the operation, but the hemiplegia has remained unimproved.

EDWIN BRAMWELL.

---

## Reviews

**VORLESUNGEN ÜBER DEN BAU DER NERVÖSEN ZENTRAL-  
ORGANE DES MENSCHEN UND DER THIERE. L.  
EDINGER. Erster Band, Siebente Auflage, 1904, Seiten 398,  
mit 268 Abbildungen. Price 12s.**

It is with pleasure we welcome this latest, the seventh, edition of Professor Edinger's well-known book, more especially as it appears in other form, and has thereby gained in many ways without losing any of the worth of its many points of excellence.

The volume under review is but half of the planned work, and only deals with the nervous system of the mammalia, but especially of man. The second volume will treat of the comparative anatomy of the brains of the lower vertebrates, but unhappily some time must elapse before its publication, as the increased interest and activity in the subject of late years will necessitate a large amount

of work, and require much time to collect and correlate the results of the various anatomists who have devoted their attention to it. It may at once be said that the volume under review is largely remodelled from the second part of the author's well-known text-book, and much increased in size. It contains 170 more pages than its predecessor, and the number of illustrations is almost doubled. The method of treatment remains on the whole unaltered, and the chapters are still in the form of lectures, which allows the author more latitude than the formal paragraphs of the usual text-book.

It is evident that Edinger's primary aim is to furnish a suitable and practical hand-book to those who need or seek such an acquaintance with the structure of the human nervous system as is a necessary basis to clinical or physiological knowledge. Further, in this edition topographical diagnosis receives careful consideration, as there is appended to the description of the anatomy of each region a careful enumeration of the symptoms associated with a lesion of any assumed part of it. These pages, to which in no small part the increase in bulk of the volume is due, cannot fail to be of the greatest service to those who attempt to study the fundamentals of local diagnosis, or to gain an insight into the correlation of structure and function in the nervous system. This attempt is fuller than that in any other text-book on the anatomy of the central nervous system known to the reviewer, and places this volume in an entirely different category.

The description of the finer anatomy of cell and fibre is fuller than heretofore. The author still tacitly accepts the neurone theory as a conception which has been remarkably fertile in both anatomy and pathology, and at present well-nigh indispensable in the study of clinical facts.

A new feature is the more detailed description of the course and connections of the smaller spinal and cerebral tracts which have only recently become accurately known through experimental and pathological researches. Thus the anatomy of the spinal cord is much more thoroughly and completely described than in the previous editions, and well illustrated by series of sections.

This edition was certainly desirable in the present state of our knowledge.

The other various chapters of the volume, too, have been further elaborated and brought up to date, and in this connection it is with pleasure we see that German work is not alone considered, nor the subject of knowledge treated as a national perquisite.

Finally, the illustrations deserve a word of praise. Nearly 150 new ones have been added, and many of the old ones improved. They include several lucid diagrams, besides evidently accurate drawings from preparations. In many instances the happy idea

of sketching-in in projection the parts neighbouring the section drawn is adopted, which enables a true and comprehensive conception to be formed of the relations and topography of the various structures.

The printing and paper are all that can be desired, and the author's clear and collected style is in marked contrast to that of many German writers.

GORDON HOLMES.

**THE PSYCHOPATHOLOGY OF EVERY-DAY LIFE.** Prof. SIGM. FREUD. Berlin, S. Karger, 1904, pp. 92. Price 3s.

THIS brochure is written on a most interesting subject, about which even the man in the street could say something. It is an investigation of the causes of such common occurrences as the forgetting of a name, a mistake in reading, writing, or action, and other things of a like trivial character. It is reprinted from the *Monatsschrift für Psychiatrie und Neurologie*.

The first question to which the author directs attention is the forgetting of proper names. The usual explanation that proper names being so seldom used are less easily recalled is not considered sufficient. He thinks there are quite definite reasons in each instance, if these are looked for with sufficient care. He gives as an example the name of a well-known painter which, on a certain occasion, he could not recall. In this instance he believes there was a disturbing effect produced by a subject he had in his mind and about which he had been conversing just before he wished to mention this name. In connection with this immediately preceding subject of conversation there were certain facts which he had to exercise some self-restraint not to talk about, and he thinks that this deliberate effort to repress or forget certain words and phrases which came into his mind, and which were circuitously related to the painter's name, had a direct influence on his inability to recall this name. Instead of the right one, two other names persisted in presenting themselves, though he knew well enough that they were wrong. He further endeavours to prove how these names presented themselves, and to his own satisfaction shows their relation to the preceding conversation and its attendant emotions. It would occupy too much space to give his account in detail, but it is very doubtful if he will succeed in convincing anyone but himself that he is right. According to his analysis of the condition there are three factors concerned; a certain disposition to forget the particular name, a shortly previous effort of repression, and the possibility of an external association between the name and the repressive effort. The two last are those chiefly concerned, he thinks.

He next proceeds to an analysis of mistakes in speaking, reading and writing, and comes to the conclusion that in nearly all such cases there is a definite disturbing influence outside the intended action, but related to it by some process of association. This influence is usually due to some associated painful emotion. Several examples of each of these mistakes are given with his explanations of them on the above supposition. In some cases these may be correct, but in most they seem to be rather far fetched, and a simpler and more probable one can be supplied. In the case of reading, for example, this simpler explanation can be supplied by Erdmann and Dodge's investigations into the mechanism of this process.

The forgetting of certain experiences and intentions are in like manner attributed to some feeling of pain or unpleasantness associated with them. It must be confessed that in some of the examples he gives the author has been hard put to it to demonstrate this relation. Mistakes in action are referred to the same category of causes.

Though one may not agree with the author's main conclusion, it cannot be denied that the subject is a most fascinating one and is worthy of further psychological analysis.

JAS. MIDDLEMASS.

## Bibliography

### ANATOMY

- COGHILL. Recent Studies on the Finer Structure of the Nerve Cell. *Journ. Comp. Neurol. and Psychol.*, April 1904, p. 171.
- MARINESCO. Recherches sur la structure de la partie fibrillaire des cellules nerveuses à l'état normal et pathologique. *Rev. Neurol.*, mai 15, 1904, p. 405.
- G. ELLIOT SMITH. A Preliminary Note on an Aberrant Circumolivary Bundle springing from the Left Pyramidal Tract. *Rev. Neurol. and Psychiat.*, May 1904, p. 377.
- BIKELES. Einige Thesen betreffend den Anordnungstypus der motorischen Zellen auf der Ursprungshöhe der Extremitäten-nerven. *Neurol. Centralbl.*, Mai 1, 1904, S. 386.
- MINGAZZINI. Experimentelle und pathologisch-anatomische Untersuchungen über den Verlauf einiger Bahnen des Zentralnervensystems. *Monatsschr. f. Psychiat. u. Neurol.*, April 1904, S. 256 u. S. 335.
- STERZI. Die Blutgefäße des Rückenmarks. Bergmann, Wiesbaden, 1904, M. 18.60.
- AGABABOW. Über die Nerven der Sclera. *Arch. f. mikro. Anat.*, April 1904, S. 701.
- WILBRAND und SAENGER. Die Neurologie des Auges. Bd. III. Anatomie und Physiologie der opt. Bahnen und Centren. Bergmann, Wiesbaden, 1904, M. 18.60.

### COMPARATIVE ANATOMY—

- C. W. PRENTISS. The Nervous Structures in the Palate of the Frog; the Peripheral Networks and the Nature of their Cells and Fibres. *Journ. Comp. Neurol. and Psychol.*, April 1904, p. 93.

G. ROMERO. Ricerche sulle terminazioni nervose nei muscoli pellicciali dorsal, della *Talpa romana* Oldf. Thom. *Bibliographie Anat.*, T. xlii., f. 2, 1904, p. 53.

### PHYSIOLOGY

POLI. Sulla funzione centrifuga delle radici posteriori della midolla spinale (Cont. e fine). *Ann. di Freniat.*, Vol. xiv., F. 1, 1904, p. 1.

BARD. Des chiasmata optique, acoustique et vestibulaire, uniformité fonctionnelle, normale et pathologique des centres de la vue, de l'ouïe et de l'équilibre. *Semaine méd.*, mai 4, 1904, p. 137.

GIULIO ANDREA PARI. Sulla tendenza delle oscillazioni automatiche dell'eccitabilità dei centri nervosi a sincronizzarsi con gli stimoli. *Sperimentale*, Anno lviii., f. ii., 1904, p. 297.

A. D. WALLER. Ueber die negative Schwankung und den elektrotonischen Quotient  $\frac{A}{K}$  des markhaltigen Warmblüternerven. *Zentralbl. f. Physiol.*, April 23, 1904, S. 33.

GENTILE. Influence de l'électricité sur le pouls cérébral. *Ann. d'Electrobiol. et de Radiol.*, Vol. vi., No. 5, p. 553.

BREUKINK. Ueber Ermüdungskurven bei Gesunden, Neurosen und Psychosen. *Monatsschr. f. Psychiat. u. Neurol.*, April 1904, S. 318.

T. R. ELLIOTT. On the Innervation of the Ileo-ocolic Sphincter. *Journ. of Physiol.*, Vol. xxxi., No. 2, 1904, p. 157.

BIELSCHOWSKY und POLLACK. Zur Kenntniss der Innervation des Säugthierauges. *Neurol. Centralbl.*, mai 1904, S. 387.

ROBERT M. YERKES. Inhibition and Reinforcement of Reaction in the Frog (*Rana clamitans*). *Journ. Comp. Neurol. and Psychol.*, April 1904, p. 124.

JENKINS and CARLSON. Physiological Evidence of the Fluidity of the Conducting Substance in the Pedal Nerves of the Slug (*Ariolimax columbianus*). *Journ. Comp. Neurol. and Psychol.*, April 1904, p. 85.

RAYMOND PEARL. On the Behaviour and Reactions of *Limulus* in Early Stages of its Development. *Journ. Comp. Neurol. and Psychol.*, April 1904, p. 133.

### PATHOLOGY

DURANTE. Régénération autogène chez l'homme et la théorie du Neurone. *Journ. de Neurol.*, avril 20, 1904, p. 147.

G. C. LOW and F. WALKER MOTT. The Examination of the Tissues of the Case of Sleeping Sickness in a European. *Brit. Med. Journ.*, April 30, 1904, p. 1000.

BABONNEIX. Nouvelles recherches sur les paralysies diphtériques. Thèse. Rousset, Paris, 1904.

LAIGNEL-LAVASTINE. Les syndromes solaires expérimentaux. *Journ. de Neurol.*, avril 20, 1904, p. 141.

PAUL MANASSE. A Contribution to the Pathological Anatomy of the Internal Ear and Auditory Nerve. *Arch. of Otol.*, April 1904, p. 93.

### PSYCHOLOGY

LOSSKY. Die Grundlehren der Psychologie vom Standpunkte des Voluntarismus. J. A. Barth, Leipzig, 1904, M. 6.

DUMAZ. Psychologie de Jeanne d'Arc. *Ann. méd.-psychol.*, mai-juin 1904 p. 353.

C. L. HERRICK. The Beginnings of Social Reaction in Man and Lower Animals. *Journ. Comp. Neurol. and Psychol.*, April 1904, p. 118.

ARNETT. The Soul. A Study of Past and Present Beliefs. *Am. Journ. Psychol.*, April 1904, p. 121.

SPEARMAN. General Intelligence objectively Determined and Measured. *Am. Journ. Psychol.*, April 1904, p. 201.

DAVIDSON. Professor Bain's Philosophy. *Mind*, April 1904, p. 161.

M'TAGGART. Hegel's Treatment of the Categories of Quantity. *Mind*, April 1904, p. 180.

ADOLF HOPPE. Leib und Seele. *Centralbl. f. Nervenl. u. Psychiat.*, April 15, 1904, S. 284.

- RUSSELL. Meinong's Theory of Complexes and Assumptions. *Mind*, April 1904, p. 204.  
 UNDERHILL. The Use and Abuse of Final Causes. *Mind*, April 1904, p. 220.  
 BENTLEY. The Psychological Meaning of Clearness. *Mind*, April 1904, p. 242.

## CLINICAL NEUROLOGY AND PSYCHIATRY

### GENERAL—

- SMITH ELY JELLIFFE. Some Notes on Dispensary Work in Nervous and Mental Diseases. *Journ. Nerv. and Ment. Dis.*, May 1904, p. 309.  
 JUDSON S. BURY. An Address on Trauma in Relation to Disease of the Nervous System. *Brit. Med. Journ.*, April 30, 1904, p. 997.  
 DURVILLE. Pour faire le diagnostic des maladies par l'examen des centres nerveux. Malverge, Paris, 1904, 1 fr.  
 VERAGUTH. Kultur und Nervensystem. Schulthess & Co., Zürich, 1904, M. 1.  
 SIMEON SNELL. Eyestrain as a Cause of Headache and other Neuroses. *Lancet*, April 30, 1904, p. 1181.  
 BRESLER. Erbsyphilis und Nervensystem. Hirzel, Leipzig, 1904, M. 2.  
 THEO. DILLER. Arterio-sclerosis of the Central Nervous System, with Report of Five Cases. *N. Y. Med. Journ.*, May 7, 1904, p. 879.  
 F. W. LANGDON. Cardio-vascular and Blood States as Factors in Nervous and Mental Disease. *Indiana Med. Journ.*, May 1904.

### MUSCLES—

- MINGAZZINI e PERUSINI. La sindrome miotonica. Miotonia congenita, miotonia acquisita e stati affini. *Riv. di Patolog. nerv. e ment.*, Vol. ix., f. 4, 1904, p. 153.  
 BRASCH. Über eine besondere Form der familiären neurotischen Muskelatrophie (Dejerine-Sottas). *Deutsche Ztschr. f. Nervenheilk.*, Bd. xxvi., H. 3, 1903, S. 302.

### PERIPHERAL NERVES—

- F. W. LANGDON. Multiple Neuritis. A Clinical Lecture. *Alienist and Neurologist*, May 1904.  
 EHRET. Weitere Beiträge zur Lehre der Skoliose nach Ischias. *Mitt. aus d. Grenz. d. Med. u. Chir.*, April 1904, S. 53.

### SPINAL CORD—

- Tabes.**—HAGELSTAM. Über Tabes und Taboparalyse im Kindes- und Entwicklungsalter. *Deutsche Ztschr. f. Nervenheilk.*, Bd. xxvi., H. 3, 1904, S. 268.  
 GORDON. Juvenile Tabes. *N. Y. Med. Journ.*, May 7, 1904, p. 872.  
 VASCHIDE et ROUSSEAU. Contribution expérimentale à la psychologie de la sensibilité tactile tabétique. Colin, Paris, 1904.  
 RODIER et CAPDEPONT. Mal perforant buccal chez une femme ataxique. *Journ. des mal. cutan. et syphilit.*, mars 1904, p. 161.  
 KLIPPEL et LEFAS. Eosinophile dans le Tabes. *Soc. de Biol.*, avril 4, 1904, p. 461.  
 ANDRÉ LÉRI. Cécité et Tabes. *Thèse de Paris*, Ruoff, 1904.  
 CHARLES W. BURR. The Influence of Fever on the Pains of Locomotor Ataxia. *Journ. Nerv. and Ment. Dis.*, May 1904, p. 320.  
 OLTAMARE. Tabes d'origine syphilitique guéri par le traitement mercuriel intensif et prolongé. *Journ. des mal. cutan. et syphilit.*, mars 1904, p. 169.  
**Spinal Tumour.**—V. RAD. Kasuistischer Beitrag zur Lehre von den Tumoren des Cervikalmarks und der Medulla oblongata. *Deutsche Ztschr. f. Nervenheilk.*, Bd. xxvi., H. 3, 1904, S. 293.  
**Syringomyelia.**—SILAS LINDQUIST. Ett fall af syringomyeli med utpräglade artropatier. *Upsala Förhand.*, April 27, 1904.  
**Multiple Sclerosis.**—DINKLER. Zur Kasuistik der multiplen Herdsklerose des Gehirns und Rückenmarks. *Deutsche Ztschr. f. Nervenheilk.*, Bd. xxvi., H. 3, 1904, S. 238.



- Meningitis.**—RAMSAY HUNT. Acute Infectious Osteomyelitis of the Spine and Acute Suppurative Perimeningitis. *Med. Rec.*, April 23, 1904, p. 641.
- Myelitis.**—DINKLER. Über akute Myelitis transversa. *Deutsche Ztschr. f. Nervenheilk.*, Bd. xxvi., H. 3, 1904, S. 248.
- SCHAEFFER. Aetiologische Betrachtungen über einen Fall von Myelitis transversa acuta infectiosa postpuerperalis e Parametritide abscedente perforante. *München. med. Wchnschr.*, Mai 17, 1904, S. 875.
- Spastic Paraplegia.**—JEFFREYS WOOD. Spastic Spinal Paralysis. *Intercolonial Med. Journ. of Australasia*, Vol. ix., No. 3, 1904, p. 138.
- Trauma.**—THEO. WEISENBURG. A Report of Three Cases of Injury to the Lower Spinal Cord and Cauda Equina. *Am. Journ. Med. Sc.*, April 1904, p. 768.
- Cytodiagnosis.**—FRENKEL. Zur Cytodiagnose bei Tabes dorsalis. *Monatsschr. f. Psychiat. u. Neurol.*, Bd. xv., H. 5, 1904, S. 390.
- FUNK. Einige Ergebnisse der Untersuchung des Liquor cerebrospinalis bei Luetischen. *Arch. f. Dermat. u. Syph.*, Bd. lxi., H. 3, 1904, S. 341.

#### BRAIN AND MENBRANES—

- Meningitis.**—VOISIN. Les méninges au cours infections aiguës de l'appareil respiratoire. *Rev. mens. des mal. de l'enfance*, mai 1904, p. 193.
- Haemorrhage.**—HOWELL EVANS. The Relation of Certain Extra- and Intra-Cranial Haemorrhages in the New-Born. *Brit. Journ. Child. Dis.*, May 1904, p. 207.
- Sinusthromboses.**—SACHS. Zur Diagnostik der Sinusthrombose. *Arch. f. Ohrenheilk.*, Bd. lxi., H. 3 u. 4, 1904, S. 176.
- STENGER. Die otitische Hirnsinusthrombose nach den in der Ohrenklinik der Charité in den Jahren 1899-1901 gesammelten Beobachtungen. *Arch. f. Ohrenheilk.*, Bd. lxi., H. 3 u. 4, 1904, S. 289.
- LEWIS MARSHALL and F. H. JACOB. Pseudobulbar Paralysis due to Bilateral Tuberculous Tumours. *Brit. Journ. Child. Dis.*, May 1904, p. 199.
- Cerebral Tumours.**—SUTHERLAND and EARDLEY HOLLAND. A Case of Glioma of the Pons. *Brit. Journ. Child. Dis.*, May 1904, p. 203.
- CHAFEY. A Case of Endothelioma of the Brain with Chronic Hydrocephalus. *Brit. Journ. Child. Dis.*, May 1904, p. 195.
- ARTHUR CONKLIN BRUSH. Report of Two Cases of Meningeal Tumor treated by Ligature of their Vessels. *Journ. Nerv. and Ment. Dis.*, May 1904, p. 318.
- McKENNAN. Two Tumors of the Brain. *Journ. Nerv. and Ment. Dis.*, May 1904, p. 301.
- Brain Abscess.**—FAUVEL. Contribution à l'étude des abcès cérébraux d'origine otitique. *Thèse*. Michalon, Paris, 1904, 2 fr.
- Brain Syphilis.**—HIRSCHL. Gehirn und Syphilis. *Wien. klin. Wchnschr.*, April 23, 1904, S. 465.
- General Paralysis.**—AUDIFFRENT. Quelques mots sur la paralysie générale. *Arch. d'anthrop. crim.*, avril 15, 1904, p. 257.
- SOUKHANOFF. Contribution à l'étude des anesthésies des organes dans la paralysie générale. *Rev. Neurol.*, avril 30, 1904, p. 365.
- SERRIGNY. La paralysie générale dans la Suisse romande. Étude statistique comparative sur la rareté de cette affection dans certaines contrées, spécialement dans le canton de Fribourg. *Ann. méd.-psychol.*, mai-juin 1904, p. 377.
- Brain Injury.**—NIMIER. Blessures du crâne et de l'encéphale par coup de feu. F. Alcan, Paris, 1904, 15 fr.

#### GENERAL AND FUNCTIONAL DISEASES—

- Paralysis Agitans.**—BURZIO. Ricerche sulla anatomia patologica del morbo di Parkinson (Cont. e fine). *Ann. di Freniat.*, Vol. xiv., F. 1, 1904, p. 34.
- Epilepsy.**—ALDREN TURNER. A Clinical Lecture on the Prognosis and Curability of Epilepsy. *Clin. Journ.*, April 20, 1904, p. 9.
- BRESLER. Die Simulation von Geistesstörung und Epilepsie. Marhold, Halle, 1904, M. 6.
- BLUMHARDT. Beitrag zur diätetischen Behandlung der Epilepsie. Pietscher, Tübingen, 1904, M. 1.
- DE BÜCK. La psycholepsie de Janet et la théorie de Storch-Foerster. *Journ. de Neurol.*, mai 5, 1904, p. 161.

- KNAPP. Ein Fall von Tastklümmung und Jackson'scher Epilepsie und seine günstige Beeinflussung durch Entfernung von adenoidem Vegetationen. *Monatsschr. f. Psychiat. u. Neurol.*, April 1904, S. 263.
- Hysteria.**—BERNHEIM. Conception du mot Hystérie. Octave Doin, Paris, 1904, 1 fr. 50.
- MONÉGER. Un cas de pied bot varus équin de nature hystérique. *Arch. de méd. et pharm. mil.*, avril 1904, p. 321.
- R. BERNARD. Confusion mentale ou hystérie. *Soc. méd. des hôp.*, mars 1904, p. 214.
- JAMES W. ALLAN. A Case of "Hysteroid" Disease in the Male. *Glasgow Med. Journ.*, May 1904, p. 346.
- SERGEANT. Délire post-grippal chez un hystérique. *Soc. méd. des hôp.*, mars 17, 1904, p. 269.
- HIRSCH. Hysterische Blindheit. *Prag. med. Wchnschr.*, April 14, 1904, S. 184.
- LEWONTIN. Ueber künstliche Hautgangrän bei Hysterischen. Günther, Berlin, 1904, M.—60.
- Neurasthenia.**—FRIEDMANN. Ueber neurasthenische Melancholie. *Monatsschr. f. Psychiat. u. Neurol.*, April 1904, S. 301.
- FRIEDMANN. Ueber neurasthenische Melancholie. *Monatsschr. f. Psychiat. u. Neurol.*, Bd. xv., H. 5, 1904, S. 368.
- DUBOIS. Troubles de la sensibilité dans les états neurasthéniques et mélancoliques. *Journ. de Neurol.*, mai 5, 1904, p. 174.
- Neuralgia.**—VINCENT. La névralgie occipitale dans les angines vulgaires. *Presse méd.*, avril 30, 1904, p. 273.
- Writer's Cramp.**—ZABLUDOWSKI. Zur Prophylaxe und Therapie des Schreib- und Musik-krampfes. *Prag. med. Wchnschr.*, April 21 u. 28, 1904.
- Kopftetanus.**—GROBER. Ein Fall von Kopftetanus. *Mitt. aus d. Grenz. d. Med. u. Chir.*, April 1904, S. 40.
- Hydrophobia.**—A. NEGRI. I risultati delle nuove ricerche sull'eziologia della rabbia. Sperimentale, Anno. lviii., f. ii., 1904, p. 273.

#### TROPIC DISEASES.—

- BONNEFANT. Du rôle étiologique de la tuberculose dans l'asphyxie locale et la gangrène symétrique des extrémités (syndrome de M. Raynaud). Maloine, Paris, 1904.
- LANNOIS et LANÇON. Trophœdème hystérique. Poinat, Lyon, 1904.
- GANDOIS. De la Gangrène symétrique des Extrémités "type Raynaud." *Thèse de Paris*, Jouve, 1904.
- VALENTIN CALONNE. Associations pathologiques dans la Maladie de Raynaud. *Thèse de Paris*, Rousset, 1904.

#### ALCOHOL, ETC.—

- BUNGE. Alkoholvergiftung und Degeneration. Barth, Leipzig, 1904, M.—40.
- NONNE. Stellung und Aufgaben des Arztes in der Behandlung des Alkoholismus. Ueber Trinkerheilstätten. Fischer, Jena, 1904, M. 1.50.
- PFAFF. Die Alkoholfrage vom ärztlichen Standpunkt. Pietzcker, Tübingen, 1904, M.—80.
- ABDERHALDEN. Bibliographie der gesamten wissenschaftlichen Literatur über den Alkohol und den Alkoholismus. Urban & Schwarzenberg, Berlin, 1904, M. 20.
- C. A. McBRIDE. The Treatment of Inebriety by Atropine. *Brit. Med. Journ.*, April 30, 1904, p. 1006.
- BACKWELL FENN. The So-called "Gold Cure" of Inebriety. *Brit. Med. Journ.*, April 30, 1904, p. 1008.

#### MENTAL DISEASES.—

- RONCORONI. Le basi di una classificazione delle malattie mentali. *Ann. di Freniat.*, Vol. xiv., F. 1, 1904, p. 49.
- FÖRSTERLING. Ein Fall von Motilitätspsychose mit vorwiegender Beteiligung der Sprache. *Monatsschr. f. Psychiat. u. Neurol.*, April 1904, S. 282.
- MAX FISCHER. Schwangerschaft und Diebstahl. *Allg. Ztschr. f. Psychiat.*, Bd. lxi., H. 3, 1904, S. 312.
- NÄCKE. Über den Wert der sogenannten "Kurven-Psychiatrie." *Allg. Ztschr. f. Psychiat.*, Bd. lxi., H. 3, 1904, S. 280.
- FLÜGGE. Über das Bewahrungshaus in Düren. *Allg. Ztschr. f. Psychiat.*, Bd. lxi., H. 3, 1904, S. 260.

- FUCHS. Ungewöhnlicher Verlauf bei "Katatonie." *Allg. Ztschr. f. Psychiat.*, Bd. lxi., H. 3, 1904, S. 241.
- TILING. Individuelle Geistesartung und Geistesstörung. Bergmann, Wiesbaden, 1904, M. 2.
- DORNBLÜTH. Kompendium der Psychiatrie für Studierende und Ärzte. Veit & Co., Leipzig, 1904, M. 5.
- COCHY MONCAN. Contribution à l'étude des stigmates de la criminalité. L'œil et la vision des criminels. *Thèse de Paris*, mars 1904.
- DUHEM. Contribution à l'étude de la folie chez les spirites. Thèse. Steinheil, Paris, 1904.
- SCHOTT. Beitrag zur Lehre von der sogenannten originären Paranoia. *Monatschr. f. Psychiat. u. Neurol.*, Bd. xv., H. 5, 1904, S. 321.
- BULLARD. Contribution to the Etiology of Idiocy and Imbecility. *Boston Med. and Sur. Journ.*, May 5, 1904, p. 471.
- MACPHERSON. Variation in its Relation to the Origin of Insanity and the Allied Neuroses (II.). *Edin. Med. Journ.*, May 1904, p. 416.
- SHUTTLEWORTH. Degeneracy, Physical, Mental, and Moral. *Med. Press and Circ.*, May 18, 1904, p. 525.
- DEWEY. A Case of Circular Insanity studied from Clinical, Differential, and Forensic Standpoints. *Journ. Am. Med. Assoc.*, April and May 1904, pp. 1127 and 1197.
- Forensic.**—SOMMER. Kriminalpsychologie und strafrechtliche Psychopathologie auf naturwissenschaftlicher Grundlage. Barth, Leipzig, 1904, M. 10.
- BATTY TUKE and HOWDEN. The Relations of the Insanities to Criminal Responsibility and Civil Capacity (V.). *Edin. Med. Journ.*, May 1904, p. 409.
- Treatment.**—DUBOIS. Les Psychonevroses et leur traitement moral. Masson et Cie., Paris, 1904, 7 fr.
- P. HOSPITAL. Regrettable insuffisance de renseignements sur les malades internés dans les asiles d'aliénés, soit volontairement, soit d'office, et le moyen d'y remédier. *Ann. méd.-psychol.*, mai-juin 1904, p. 403.
- Cranial Nerves.**—V. HIPPEL. Neuere Untersuchungen zur Physiologie und Pathologie der Pupillenphänomene. *München. med. Wchnschr.*, April 19, S. 692.
- BÄRTELS. Ueber die Beziehungen zwischen Trigeminusneuralgie und der residuierenden Hornhauterosion. *München. med. Wchnschr.*, April 28, 1904, S. 746.
- V. BECHTEREW. Ueber die Beteiligung des Musculus orbicularis oculi bei corticalen und subcorticalen Facialisparalysen. *Centralbl. f. Nervenheilk. u. Psychiat.*, Mai 15, 1904, S. 305.
- CUTHBERT WALLACE. A Case of Attempted Division of the Eighth Nerve within the Skull for the Relief of Tinnitus. *Lancet*, April 30, 1904, p. 1192.

## REFLEXES—

- STANLEY BARNES. The Diagnostic Value of the Planter Reflex. *Rev. Neurol. and Psychiat.*, May 1904, p. 345.
- V. BECHTEREW. Ueber eine eigenthümliche Reflexerscheinung im Gebiete der Extremitäten bei centralen organischen Paralysen. *Neurol. Centralbl.*, Mai 16, 1904, S. 434.
- AUGUSTUS ESHNER. Some Graphic Observations on Ankle Clonus. *Journ. Am. Med. Assoc.*, May 1904, p. 1202.
- PANDY. Die Kraft der Sehnenreflexe und die Veränderung derselben bei der Hemiplegie. *Neurol. Centralbl.*, Mai 16, 1904, S. 449.
- STEINER. Über den Kremasterreflex und die Superposition von Reflexen. *Deutsche Ztschr. f. Nervenheilk.*, Bd. xxvi., H. 3, 1904, S. 285.

## MISCELLANEOUS SYMPTOMS—

- GRASSET. De la déviation en sens opposé de la tête et des yeux ; paralysie d'un oculo-gyre et contracture du céphalogyre homonyme. *Semaine méd.*, mai 18, 1904, p. 153.
- DANESCHI e SERGI. Il senso muscolare nelle lesioni del cervello. Firenze, 1904.
- HERMANN H. HOPPE. A Clinical and Pathological Contribution to the Study of the Central Localization of the Sensory Tracts. *Journ. Nerv. and Ment. Dis.*, May 1904, p. 231.
- E. NOBLE SMITH. Abnormal Muscular Contractions. *Lancet*, May 7, 1904, p. 1269.
- GRASSET. Un cas de voix eunucoïde, datant de l'ictus, dans l'hémiplégie cérébrale (paralysie du crico-thyroïdien). *Journ. de Neurol.*, avril 20, 1904, p. 151.

- KORNFELD. Verbrechen und Geistesstörung im Lichte der altbiblischen Tradition. Marhold, Halle, 1904, M.—80.
- WOLFF. Klinische und kritische Beiträge zur Lehre von den Sprachstörungen. Veit & Co., Leipzig, 1904, M. 2.40.
- LARRIVE. Un cas de retard de la parole par malformation anatomique chez un arriéré épileptique. Tentative de traitement chirurgical. *Journ. de Neurol.*, mai 5, 1904, p. 176.
- HESS. Retrograde Amnesie nach Strangulationsversuch und nach Kopftetanus. *Monatsschr. f. Psychiat. u. Neurol.*, April 1904, S. 241.
- J. JAMESON EVANS. Neuro-retinitis in Chlorosis. *Lancet*, May 14, 1904, p. 1345.
- MIBELLI. Ueber einige Fälle von Neurodermatitis chronica linearis. *Monatsschr. f. prakt. Dermat.*, Bd. 38, H. 9, 1904, S. 413.
- DOERNBERGER. Pneumonie, Meningismus und Aphasie. *München. med. Wchnschr.*, Mai 10, 1904, S. 833.

#### TREATMENT—

- BULLARD. Care of Epileptics in Private Practice. *Pediatrics*, April 1904, p. 158.
- BELLINI. La bioplastina nell' uso terapeutico di alcune psicopatie. Torino, 1904.
- ILBERG. Irrenanstalten, Idioten und Epileptikeranstalten mit besond. Berücksicht des Arztes in denselben. Fischer, Jena, 1904, M. 2.
- SERRIGNY. A propos de l'alimentation forcée chez les aliénés par la voie buccale. Mandrin pour faciliter l'introduction de la sonde œsophagienne. *Journ. de Neurol.*, avril 20, 1904, p. 155.
- MELTZER. Die staatliche Schwachsinnigenfürsorge im Königreich Sachsen. *Allg. Ztschr. f. Psychiat.*, Bd. lxi, H. 3, 1904, S. 370.
- VIDAL THORPE. A Chinese Remedy for Hydrophobia. *Lancet*, May 14, 1904, p. 1341.
- Surgical.**—SAM. MIXTER. Operation in Spinal Cord Injuries. *Ann. Surg.*, April 1904, p. 495.
- ALFRED S. GUBB. Intraspinal Cocainization. *Brit. Med. Journ.*, April 23, 1904, p. 950.
- TUFFIER. La Rachicocainisation. Naud, Paris, 1904.
- CHIPAULT. Manuel d'Orthopédie vertébrale. Maloine, Paris, 1904, 5 fr.
- A. BERT et P. VIGNARD. La topographie cranio-cérébrale simplifiée et le craniomètre de Kroenlein. *Rev. de Chir.*, avril 1904, p. 562.
- FERDINAND ALT. Operative Eingriffe in der hinteren Schädelgrube. *Wien. klin. Wchnschr.*, Mai 1904, S. 524.
- JONATHAN HUTCHINSON. Intracranial Resection of the Second Division of the Fifth Nerve for Epileptiform Neuralgia. *Med. Press and Circ.*, May 4, 1904, p. 465.
- G. MARION. La craniectomie au moyen de la scie de Gigli. *Arch. gén. de méd.*, avril 28, 1904, p. 1025.
- Electrical.**—CLARENCE WRIGHT. On the Methods of Application of High Frequency Currents. *Med. Electrol. and Radiol.*, May 1904, p. 191.
- DELHERM et LAQUERRIÈRE. L'électricité dans le traitement des accidents hystériques. *Arch. d'Electricité méd.*, avril 25, 1904, p. 288.
- BONNEFOY. Études cliniques sur l'action thérapeutique des courants de haute fréquence et de haute tension dans les maladies par ralentissement de la nutrition. *Ann. d'Electrobiol. et de Radiol.*, Vol. vi., No. 5, p. 513.

# Review of Neurology and Psychiatry

---

## Original Articles

### DISSEMINATED SCLEROSIS.

#### **An Account of the Microscopical Examination of Three Cases with some Observations on the Pathogenesis of the Disease.**

By A. F. TREDGOLD, L.R.C.P. Lond., M.R.C.S. Eng.,  
Formerly London County Council Research Scholar in Neuropathology.

#### INTRODUCTION.

"DISSEMINATED," "Multiple," or "Insular" Sclerosis is an affection of the nervous system due to the presence of indiscriminate islets or patches of disease. So much has been known since the days of Cruveilhier; but although from that time much attention has been given to the subject, opinions still differ widely as to the nature of these islets and the manner in which they are produced. In this paper it is proposed to give an account of the microscopical appearances of three cases of this disease, each of which was typical of a distinct clinical variety, in the hope that they may add a little to our knowledge of this interesting condition. After this I shall briefly consider the views hitherto advanced regarding its pathogenesis.

At the outset it must be remarked that definite foci, islets or patches of sclerosis, occur in other conditions than that with which we are now dealing; thus, islets may be the result of syphilis, they may replace hæmorrhagic foci of softening, or they may be due to anomalies of development; and not only may the

naked eye appearances of such islets closely resemble those of disseminated sclerosis, but occasionally the clinical resemblance may be so striking as to deceive the most competent observers. It is probably to some extent due to this and to the additional fact that disseminated sclerosis itself occurs in several distinct clinical varieties, that much of the confusion regarding the morbid anatomy is due.

Without entering into any detailed description of the symptomatology, it may be useful to remark that the following clinical types of disseminated sclerosis are recognisable:—

1. *The classical type*, in which the predominant symptoms are tremor, nystagmus, and alteration of speech.
2. *The spastic paraplegic type*, in which for a considerable period the clinical picture is indicative only of a lateral sclerosis. (Case 1 was of this nature.)
3. *The combined lateral and posterior sclerosis type*, in which spastic paraplegia is accompanied by sensory changes.
4. *The transverse myelitis type*. (Case 2 was of this nature.)
5. *The cerebellar type*, characterised by the cerebellar gait and often accompanied by headache, giddiness, vomiting, and symptoms suggestive of cerebellar tumour. (Case 3 was of this nature.)
6. *The hemiplegic type*, in which apoplectic attacks occur followed by hemiplegia, with or without aphasia. Usually the paralysis is of a transient nature.
7. *The hysterical type*, marked by transient paralyses following nervous shock or actual injury.

So long as a case conforms to one of the types 2 to 7, and presents no other distinguishing features, diagnosis is of course extremely difficult and may be impossible; to many cases, however, beginning in one or other of these ways, there are sooner or later added other special signs which show their real nature. Amongst these special symptoms, tremor, nystagmus, and speech alteration are singularly common; but even in their absence, provided that the other forms of irregular sclerosis above referred to can be excluded, a careful examination will often reveal some peculiarity in the grouping of motor, sensory or reflex signs which could only be brought about by disseminated sclerosis. Thus, if the clinical account of the three following cases be

scrutinised, it will be seen that although their chief symptoms correspond to one or other of the above types and simulate those diseases, there were nevertheless present other signs which were not characteristic of those diseases, and which in fact could only be produced by disseminated lesions. I think the greatest difficulty of all is to distinguish between those cases of irregular lesions which are due to syphilis.

The three cases examined are typical of the 2nd, 4th and 5th of the above varieties, and I shall hope to show that in each the pathological condition is identical in kind, and differs only in the degree or stage of the morbid process and in the situation of the lesions.

## SECTION I.

### CLINICAL AND POST-MORTEM ABSTRACT OF CASES.

#### CASE No. 1. Spastic paraplegic type—8 years' duration.

##### CLINICAL.

E. J. S., a married woman, 44 years of age, was admitted into the London Hospital on Sept. 16, 1897, under the care of Dr F. J. Smith—to whom I am indebted for permission to publish the clinical notes.

*Family history.*—Patient's father was "epileptic" and died paralysed and out of his mind in an asylum, aged 45 years. Patient's mother is alive and well, age 74. All her brothers and sisters are living and in good health.

*Personal history.*—The patient was in good health until her last confinement eight years ago, when delivery was instrumental and there was a large perineal tear. Shortly afterwards she began to suffer from weakness of the legs and dribbling of urine; these got worse, but she was able to hobble about with a stick until five months before admission, since then having been bedridden. There have never been any exaggerated movements of the legs, but she states that when sitting or lying down they would frequently be "drawn up" and she would have great difficulty in putting them straight again.

*On admission.*—There was almost complete paralysis of both legs, the patient only being able to move the toes, slightly flex the knees and slightly adduct the thighs. The movements of

the face, tongue, neck and arms were quite unaffected and free from tremor. The electrical reactions of the leg muscles were sluggish but there was no qualitative change. Sensation to touch, pain, heat and cold was normal all over the body. Sense of position of the paralysed legs was also normal. There was no pain. The knee-jerks were increased and double ankle clonus was present, the other reflexes were normal. There was incontinence of urine, but the control over the rectum was unimpaired. The pupils reacted to L and A, no strabismus and no definite nystagmus, the optic discs were normal. Speech was high-pitched and mumbling, the mental condition was one of apathy with some impairment of memory for remote events.

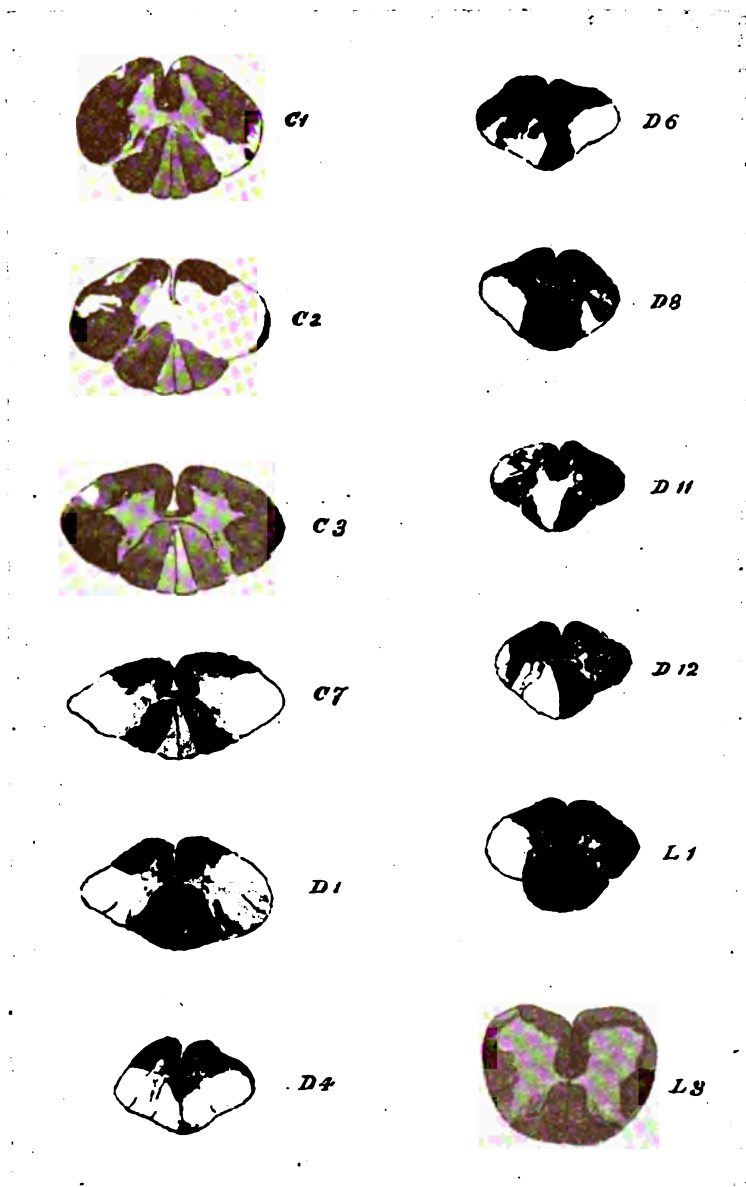
*Progress.*—There was no observable change for three weeks. She then had a rigor with rise of temperature to  $105^{\circ}$ , retention of urine and incontinence of fæces. The temperature became normal again in thirty-six hours, but it was then found that all her nervous symptoms were more intense. During the next three weeks she had three more such attacks, gradually becoming more apathetic, with pronounced nystagmus, deglutition very difficult and articulation at times impossible. After the first of these attacks the knee-jerks, which had previously been much exaggerated, were found to be normal and the ankle clonus had disappeared. After the second, pus appeared in the urine and there was persistent vomiting. She gradually lapsed into a semi-comatose state and died six weeks after admission.

#### POST-MORTEM EXAMINATION.

There was chronic cystitis and suppurative pyelitis, but the heart, lungs and other organs were healthy.

The *skull, membranes, vessels and sinuses* were normal. The *brain* weighed 40 oz., its convolutions being of average complexity but somewhat atrophied. Horizontal sections through the hemispheres revealed the presence of many islets of disease; these were almost entirely confined to that portion of the white matter adjacent to the cavities of the lateral ventricles. In size they ranged from small spherical areas of 2 to 3 mm. diameter to large vertical fusiform patches measuring 10 to 12 mm. in breadth and as much as 30 to 40 mm. in length. Some of these were grey, soft and translucent; others were yellow and





DISSEMINATED SCLEROSIS.

Sections of spinal cord drawn with the Edinger projection apparatus.  $\times 3$ . [E. J. S., Case No. 1.]

[illegible]

firm; and yet others were of intermediate consistence. In addition there were a few areas of disease, pea-like in size and shape, scattered through the white matter of the centrum ovale. All the patches were sharply defined. The *cerebellum* and *pons* were healthy. The *medulla* contained a few scattered hard islets.

*Spinal cord.*—The entire cord was small and shrunken and lay very loosely in its sheath. After hardening, horizontal sections were made every 4 mm., and it was then seen that the cord was practically riddled with sclerotic islets. These varied in size from quite small circular or wedge-shaped patches to large spindle-shaped masses running through several spinal segments. The greatest amount of sclerosis was in the thoracic region, where indeed there appeared to be almost more diseased than healthy tissue; the cervical portion was affected next and the lumbar least of all. The distribution of the patches will be seen by reference to Plate 7.

#### CASE NO. 2. Myelitis type—3 years' duration.

##### CLINICAL.

J. S., male, aged 30 years; occupation, upholsterer; admitted into the National Hospital, Queen Square, June 14th, 1899, under the care of Dr Ferrier, F.R.S., to whom I am indebted for permission to publish the clinical notes.

*Family history.*—Nil.

*Personal history.*—He was in good health until two and a half years previously, when he had "influenza"; shortly afterwards he began to notice difficulty in walking and in passing his water, also pain and stiffness in the right leg, later on extending to the left. There was also a "tight feeling" round his waist and at times double vision. A year ago he had a severe attack of vomiting, and six months ago he began to have nodding movements of the head. No syphilis.

*On admission.*—The sight was normal, pupils equal and reacted to L and A, no ocular paralysis, but nystagmus was present on lateral deviation. The discs were pale. Hearing normal; speech slow and scanning. The facial muscles were slightly weaker on the right side and the tongue deviated to the right on protrusion; the grasp of the right hand was decidedly

deficient in power. Nodding movements of the head were present as well as intention tremors in both arms and considerable inco-ordination of the legs, so that he was unable to walk without support, although no muscular wasting was apparent. Both knee-jerks were present and there was double ankle clonus and extensor response. He complained of abdominal pain resembling girdle sensation, but otherwise there was no sensory disturbance.

*Progress.*—During August he had three attacks of vomiting, but otherwise was better as regards walk, co-ordination of hands, and nodding movements of head. He was sent away to the country for two months, and was then readmitted with signs of extensive phthisis and great general muscular wasting. Knee-jerks were absent, there was complete loss of control over both sphincters, and he complained of giddiness and pain in the back. Three weeks afterwards, knee, wrist, and elbow jerks could not be obtained, there was double extensor response but no anæsthesia. The leg muscles only just reacted to the strongest faradic current applied by means of needles inserted into the muscles. He died on December 10th, 1899, there being no further change recorded in his nervous symptoms.

#### POST-MORTEM EXAMINATION.

General muscular wasting. *Heart* normal. *Lungs*, extensive tubercle in both. *Kidneys* contained caseous tubercle, pelvis inflamed and much thickened. *Bladder* in a condition of chronic inflammation.

The *skull*, *membranes*, *vessels* and *sinuses* were perfectly healthy. The *brain* was well convoluted and appeared normal externally, but on making horizontal sections the white matter of the hemispheres was found to be occupied by many small islets of disease of hard, soft and intermediate consistency similar to those in Case 1. The *cerebellum* contained several dense patches, which were strictly limited to the white matter. The *crura*, *pons* and *medulla* contained several islets as well as an irregular patch of dense sclerotic tissue surrounding the greater part of the length of the aqueduct and floor of the fourth ventricle. In the *chiasma* and *optic nerves* there were several firm islets. *Spinal cord*.—Nothing abnormal was seen externally, and the cord was of ordinary size and shape, unlike the distorted and con-



C1



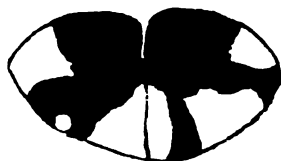
D6



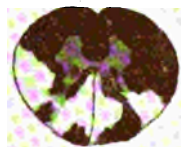
C3



D9



C4



D12



C6



L2



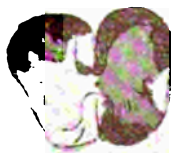
D1



LF



D3



S1

A. F. J. J. J. J.

DISSEMINATED SCLEROSIS.

Sections of spinal cord drawn with the Edinger projection apparatus.  $\times 3$ . [J. S., Case No. 2.]

1  
1  
1  
1  
1  
1

tracted one of Case No. 1. On making horizontal sections after hardening, however, a very large number of islets were found in every region. Most of these were hard and well defined, but a few were less dense and shaded so gradually into the normal tissue that it was difficult with the naked eye to make out their precise extent. (See Plate 8.)

CASE NO. 3. Cerebellar type—15 months' duration.

CLINICAL.

H. B. R., male, single; occupation, clerk; age 21 years; was admitted into the National Hospital, Queen Square, on March 27th, 1899, under the care of Dr Hughlings Jackson, F.R.S., to whom I am indebted for permission to publish the clinical notes.

*Family history.*—Father died of phthisis. Mother alive and well. No history of insanity, epilepsy or nervous disease.

*Personal history.*—He was quite well until six months ago, when he began to be troubled with giddiness and dimness of sight. His eyesight would be dim all day, and then whilst stooping down would suddenly become perfect again. Three months ago he began to have violent occipital headache and frequent vomiting, and shortly afterwards double vision. These symptoms steadily became worse, and he began to fall about whilst walking. For a month prior to his admission he has had to keep to bed.

*On admission.*—The patient was a poorly nourished, delicate looking man, complaining much of headache, and vomiting on slight movement. Taste and smell were normal. Pupils were equal and reacted to L. and A. The optic discs were rather grey and muddy with very marked scleral rings; the fields of vision were normal. There was slight convergent strabismus due to weakness of the external recti, also occasional diplopia and nystagmus on performing lateral movements. There was slight weakness of the left facial muscles, both upper and lower, and the tongue deviated to the left on protrusion. The movements of the arms were good beyond some slight clumsiness on attempting to touch the nose with the left forefinger. The movements of the legs were likewise good. There was unsteadiness on standing with the feet together, and on shutting the eyes he tended to fall backwards and to the left. No tremor. Sensation unimpaired. Reflexes

normal. No mental change. The case was considered to be probably one of cerebellar tumour.

*Progress.*—The patient improved slightly until the end of August 1899. Then his head and arms began to shake and he began to lose the power of moving his legs. The headache, giddiness and vomiting came on again, the latter being so bad that he was unable to sit up. His articulation became slow and staccato, with at times a slurring over syllables, and on again examining the eyes marked optic atrophy was found with contraction of the fields of vision. The slightest attempt to move caused pronounced tremor of the head and jaw; there was also extreme intention tremor of both arms. In the lower extremities there was marked general wasting, and although he could perform all movements whilst lying down he was unable to stand or walk. Exaggerated knee-jerks, ankle clonus and extensor response were present on both sides. On November 9th he was much worse, the legs intensely wasted, and hardly any voluntary movement of the right. Ankle clonus had disappeared, and the right knee-jerk was difficult to obtain. Some impairment of sensation below the umbilicus. On December 10th there was complete paralysis of both legs, and almost complete anæsthesia and analgesia below the umbilicus. Right knee-jerk absent, left just present. On December 28th there was complete anæsthesia as high as the ninth dorsal level with complete incontinence and absent knee-jerks. The patient gradually became weaker and died on January 9th, the nervous symptoms remaining the same except that the plantar reflexes could not be obtained for a day before death.

#### POST-MORTEM EXAMINATION.

*Heart* normal, one small caseous tubercular focus in the *lungs*. *Bladder* in a state of chronic inflammation; other organs healthy. The *skull*, *vessels* and *sinuses* were healthy. The *pia arachnoid* was slightly opaque and the convolutions were somewhat shrunken, but otherwise the brain presented no abnormal appearances externally. Horizontal sections of the *brain* revealed the presence of many small circular islets scattered indiscriminately through both hemispheres. In the *corpora quadrigemina* there were several very large areas of sclerosis; the *pons* was comparatively little affected. The *cerebellar peduncles* contained several





C3



D12



C4



L2



C6



L4



D1



L5



D6



S2

*A. F. Wood*

DISSEMINATED SCLEROSIS.

Sections of spinal cord drawn with the Edinger projection apparatus.  $\times 3$ . [H. B. R., Case No. 3.]



sclerotic areas, but there were none in the lobes of the *cerebellum*.

The upper portion of the *medulla*, with the exception of the pyramids, was almost entirely sclerosed; in the lower half also were several scattered patches, but this portion was much less affected.

*Spinal cord*.—There was considerable venous engorgement of the membranes, and in the lumbar region the dura was slightly thickened and adherent to the underlying arachnoid. The cord itself was large, swollen and tense, contrasting very strongly with the small contracted cord of Case No. 1. On making horizontal sections there was found to be scarcely any portion which was free from disease; but instead of the sharply defined hard islets characteristic of the preceding cases, in this case the patches were softer in consistence and much less definitely marked out. To the naked eye, indeed, the general appearance was more like that of a diffuse patchy myelitis. (See Plate 9.)

## SECTION II.

### MICROSCOPICAL EXAMINATION OF THE CASES.

It is desirable to state that the microscopical examination has been as complete as possible. Altogether more than 500 sections were examined, these being taken from different regions of the brain, cerebellum, optic tracts, crura, pons, medulla and spinal cord, as well as from the peripheral nerves and posterior spinal ganglia in each case. Horizontal, longitudinal and teased specimens have been prepared, and practically every modern method of staining the nervous system has been employed. I am greatly indebted to Dr F. E. Batten for kindly placing at my disposal the material from Cases 2 and 3.

I propose to describe the findings under the following heads:—

- (a) *The islets.*
- (b) *Nerve fibres.*
- (c) *Nerve cells.*
- (d) *Vessels, membranes and connective tissue.*

#### (a) THE ISLETS OF DISEASE.

Foci of disease were found in all parts of the central nervous system, although such were not all islets of sclerosis, some being

quite soft and others of an intermediate consistence. Within the medulla and cord, where they are most plentiful, their transverse section is commonly wedge-shaped, the base of the wedge being towards the periphery, and their size varies from a mere speck hardly discernible by the naked eye, to an area involving a third or even half of the entire section. They are much more common in the white than in the grey matter, but their situation is entirely unrelated to any of the conducting tracts. The islets frequently surround the fissures, and although, generally speaking, they are of indiscriminate arrangement, many sections show them to be somewhat symmetrically placed in the right and left halves. Longitudinal sections show that the islets are of considerable extent, since they may often be traced through several spinal segments, and from these sections it is apparent that their true shape is cylindrical or fusiform rather than spherical. Within the brain the islets are usually much smaller but often of the same fusiform character, and they are most plentiful in that portion of the centrum ovale adjacent to the lateral ventricles; indeed, in many cases they are in actual contact with and seem to extend inwards from the ventricular wall, precisely as do those in the cord from the fissures. Within the pons and medulla also the islets are commonly arranged around the Sylvian aqueduct or floor of the fourth ventricle.

It has already been remarked that by the naked eye it was possible to divide these islets of disease into three varieties according to their consistence, viz., *hard*, *soft* and *intermediate*; under the microscope, however, it is seen that although many islets occur having these characters, it often happens that they are all combined in one islet, in such cases the densest tissue being usually in the centre; nevertheless, the terms are convenient for descriptive purposes, and the microscopical structure of these varieties is as follows:—

*Hard islets.*—These are chiefly met with in the cord, and consist of a dense interlacement of thickened neuroglia fibrils with very few glia cells. Nerve fibres are practically absent, although occasionally a naked axis cylinder is met with. There are no products of degeneration and but few blood-vessels. The great majority of the islets present in Case No. 1 were of this nature. (See Plate 14, Fig. 4.)

*Soft islets.*—Typical patches of softening were confined to the



FIG. 1. Horizontal section of optic chiasma showing sclerotic areas (A). [J. S., Case No. 2, Carmine-Weigert.]



FIG. 2. Longitudinal section of spinal cord at level of 5th dorsal segment. A, Normal cord. B, Islets of sclerosis. [J. S., Case No. 2, Marchi-Pal.]

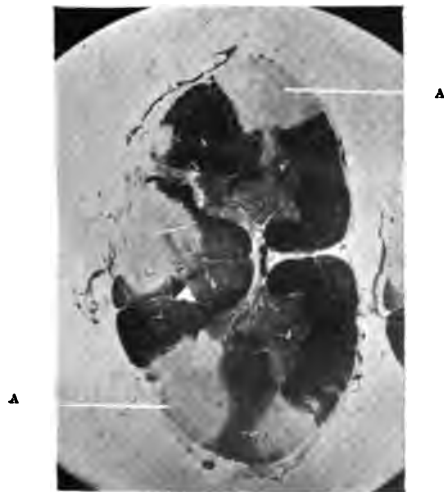


FIG. 3. Section of spinal cord at level of 4th cervical segment, showing sclerotic islets (A). [J. S., Case No. 2, Marchi-Pal.]



FIG. 4. Section of medulla, showing sclerotic islets. [J. S., Case No. 2, Marchi-Pal.]



brains; in the fresh state they were very soft, translucent, and of a gelatinoid appearance, and they hardened in Müller's fluid with extreme slowness. They usually have a well-defined margin which is often surrounded by a zone of leucocytes, and they consist of a loose reticulum containing a semi-fluid material. They are generally devoid of nerve cells or fibres; there is no proliferation of neuroglia and usually no sign of degeneration products, their vessels are often much congested.

*Intermediate islets.*—Within these, nerve fibres are found in every stage of degeneration, from cloudy swelling of the myelin sheath to complete disruption. Products of degeneration consisting of free droplets of myelin and fat, also corpuscles of Glüge are abundant. The neuroglia normally present between the nerve tubules is somewhat thickened, and has a finely granular structure, but there are no new fibres, although glia cells are often plentiful. The vessels are distended, and their lymphatics filled with leucocytes and fat-containing cells. Most of the islets in the cord of Case No. 3 were of this nature. They were also abundant in Case No. 2, but rare in Case No. 1. (See Plate 12, Fig. 11; and Plate 14, Fig. 3.)

#### (b) NERVE FIBRES.

##### 1. *Medullary Sheath.*

The condition of the myelin sheaths within the islets described was as follows:—

(a) *Normal.*—There were very few of these, and they were restricted to those islets in which neuroglial proliferation was either absent or only very slight, and in which products of degeneration were very scanty; in other words, to those islets of probable recent formation.

(b) *Cloudy swelling.*—Within the same islets a considerable number of the sheaths were swollen and cloudy in appearance; in transverse sections their concentric rings could not be seen, and in longitudinal sections their calibre was irregular. (See Plate 11, Figs. 5, 6, 8; and Plate 14, Fig. 2.)

(c) *Granular.*—Many sheaths, in addition to being swollen and varicose, had a distinctly granular appearance, and by Stroebe's method they were of a pink or lilac colour. (See Plate 12, Figs. 11, 12; and Plate 14, Figs. 1, 2.)

(d) *Fatty degeneration.* — Marchi's method of staining revealed many sheaths in a typical condition of fatty degeneration; in places the broken down myelin was becoming detached from the cylinder leading to the final change. (See Plate 12, Figs. 9, 10; and Plate 15, Fig. 5.)

(e) *Complete disappearance* of the sheath, leaving a naked axis cylinder. (See Plate 14, Fig. 3.)

## 2. *Axis Cylinder.*

By suitable methods of staining, it was found that many axis cylinders also showed degenerative changes. The earliest departure from the normal consisted in an irregular swelling, such cylinders being angular in transverse and varicose in longitudinal sections. This was followed by an alteration of staining reaction, the cylinders being lilac in colour instead of the normal dark blue when treated by Ströbe's method. Finally, in most cases fatty degeneration and disintegration of the cylinder occurred, although in some fibres the process appeared rather to be of the nature of a gradual atrophy. It is important to notice, however, that in no case was any change found in the cylinder in the absence of change in the sheath; and very often before any alteration could be detected in the former, the myelin sheath had undergone well-marked fatty degeneration, and in many instances had completely disappeared. But the sheath having been destroyed, the death of the cylinder seems to be but a question of time; since although it occasionally happens that naked axis cylinders are to be found even in quite dense islets, such an occurrence is decidedly rare, and many of these naked cylinders show pathological change on careful examination. In Case No. 1, where most of the patches were dense and old, naked cylinders were rare, but in the more recent and softer patches of Cases 2 and 3 they were decidedly more numerous. (See Plate 14, Figs. 1, 2, 3; and Plate 15, Fig. 5.)

## 3. *Peripheral Nerves.*

In the peripheral nerves of Cases 1 and 2, similar changes to those above described were found, many of the myelin sheaths being irregular in outline and cloudy or granular, others apparently disintegrating. (See Plate 12, Fig. 12; and Plate 14, Fig. 1.) I could not, however, find any naked cylinders, nor did Marchi's



method reveal fatty products of degeneration in any quantity. It seems probable that degeneration products are carried away much more quickly from the peripheral nerves than from the spinal cord. The anterior and posterior nerve roots showed similar changes. No pathological change could be detected in the nerves from Case 3. I could not detect any abnormal condition of the sheath of Schwann or its nuclei.

(c) NERVE CELLS.

*Cells of the cortex cerebri.*—Many of the cortical cells of Cases 1 and 2 were pigmented and in a condition of chronic atrophy like those of the cord, also the peri-cellular spaces were considerably dilated.

*Motor cells of the anterior cornua.*—In Case No. 1, marked changes occurred in a considerable number of those cells. No particular groups or segments were specially involved, the degenerate cells being scattered amongst healthy ones in all parts of the cord. There was no *acute* degeneration, the change being in every instance a chronic one, very similar to that which Mott and myself have described as occurring in primary degeneration of the motor tract. The chromatoplasm first loses its regular arrangement, and becomes aggregated into irregular clumps, its place being taken by a mass of yellow pigment, which stains black with Marchi's fluid. Next the nucleus loses its distinct outline, and is displaced to the periphery of the cell, the cell processes become thinner and gradually disappear, the whole cell becomes smaller, loses its characteristic shape, and is eventually represented by a small angular or rounded body with but little affinity for stain. In Case 2, fewer cells were affected, and such had not got beyond the pigmentary stage. In Case 3, none of the cells showed definite pathological change. (See Plate 13, Fig. 15; Plate 15, Fig. 6.)

*Cells of the posterior spinal ganglia.*—In Case No. 1, although the majority of the cells appeared healthy, others were in a condition of chronic degeneration like that just described. Such degeneration was found in both large and small cells in all the ganglia examined. (See Plate 15, Fig. 7.) There was practically no change in the cells of Cases 2 and 3.

It is of importance to notice that these atrophic changes are not the direct result of the presence of an islet of sclerosis, since

they frequently occur in cells entirely removed from any such islet, and indeed where sclerotic tissue does involve the grey matter it is quite common to find an ordinary proportion of perfectly normal cells. It is also of interest to note the entire absence of acute change, and the fact that cellular degeneration is by far most pronounced in the most chronic case.

#### SECONDARY DEGENERATION.

It will readily be understood that the demonstration of secondary degeneration in this disease is not an easy matter. For the haphazard distribution of the islets, and the fact that in them the process by no means affects *all* the nerve fibres simultaneously, would give rise to degenerated fibres in many tracts rather than to a limitation of the secondary change to one particular tract; and even should such change be acute, and therefore stainable by Marchi's method, the difficulty of detection amid all the scattered islets present will be obvious. And should the case under examination be a more chronic one, as is much more likely, and the products of secondary degeneration cleared away and replaced by interstitial sclerosis, it will readily be conceded by anyone who has had experience of neuropathological research that the difficulty of distinguishing between the interstitial sclerosis and islets of disease will be even greater. Consequently it is not altogether surprising to hear that in this disease secondary degeneration does not occur, a statement which I believe to be entirely erroneous.

In Case No. 1, which was of eight years' duration, there was but little secondary Marchi change, but there was very considerable interstitial sclerosis, quite distinguishable from the islets, in both the lateral and posterior columns. In the lateral columns such change could be plainly traced as far downwards as the second lumbar segment, whilst the posterior columns were sclerosed upwards from the fourth thoracic segment, evidently as a result of the very large islet occurring at that level.

In Case No. 2, interstitial sclerosis, with diminution in the number of nerve fibres, was also present in the lateral and posterior columns; and in this case, which, it will be remembered, was more acute than Case No. 1, there also occurred in all parts of the cord a considerable number of nerve fibres

undergoing ascending and descending degeneration and staining by Marchi's method.

In Case No. 3 there was much scattered Marchi degeneration in all parts of the cord, but owing to the amount of change present no definite ascending and descending degeneration could be made out.

#### (d) VESSELS, MEMBRANES, AND CONNECTIVE TISSUE.

In every case the *vessels* of the central nervous system were greatly distended with blood, and the peri-vascular lymphatics dilated and filled with leucocytes; where disintegration was proceeding abundant products of degeneration were also found in the lymphatics and adjacent tissues. In no instance could I find any trace of hæmorrhage, thrombosis or embolus.

In the acute case (No. 3) the *vessel walls* showed no pathological change whatever, but in Cases 1 and 2 there was slight fibroid or hyaline thickening of the media and adventitia of several, chiefly the smaller ones. These changes, however, were not confined to the vessels within the islets, but occurred in those amidst healthy tissue. The intima was in every case normal.

Within the denser form of islets thickening of the connective tissue septa occurred, and in Cases 1 and 2 there was slight thickening of the investing membrane of the cord; otherwise no definite pathological change could be detected in the membranes of either brain or cord beyond an increased vascularity.

Special stains and very fine paraffin sections were used to discover the presence of micro-organisms, but with negative result.

### SECTION III.

#### OBSERVATIONS UPON THE PATHOGENESIS.

Having now described the microscopical appearances of these three cases, we may consider their interpretation and what are the rôles played respectively by the neuroglia, the blood-vessels, and the nerve fibres in the *tout ensemble* of the disease. Regarding this, there is still much difference of opinion, and three views, which may be termed the *Primary sclerotic*, the *Vascular*, and the *Parenchymatous* are current.

The *Primary sclerotic* view maintains that the primary and essential change is a proliferation of the neuroglia, which, apparently by a process of strangulation, brings about degeneration of the nerve tubules. It is contended by Strümpell and other upholders of this view that such neuroglial overgrowth is the result of inherited tendencies.

According to the *Vascular* theory, the initial change consists in a structural alteration of the vessel walls, whereby the circulation is so far impeded as to cause either destruction of the nerve tubules, with subsequent overgrowth of neuroglia in and around the damaged area, or neuroglial hyperplasia with subsequent degeneration of nerve tubules.

The *Parenchymatous* theory contends that the morbid process involves first of all the myelin substance of the nerve tubules; that this is independent of structural vascular changes, although it may possibly be due to a circulating "toxin"; and that the proliferation of neuroglia is purely a secondary result precisely similar to that which occurs in the systemic degenerations of the cord.

Now it is unquestionable that islets of sclerosis may result from pathological processes in accordance with each of these theories. I have examined several cases where definite islets occurred in the brain, in which I could come to no other conclusion than that they were due to a primary overgrowth of neuroglia. In two cases, in addition to the islets, there were dense nodular excrescences of almost pure glia tissue projecting from the walls of the lateral ventricles (*sclérose tubéreuse*). In another case there was a thick band of neuroglia capping the brain cortex in certain areas. These cases, however, were not disseminated sclerosis; all the patients were epileptic and mentally defective, and there were no symptoms by which the sclerotic areas could have been definitely diagnosed during life. I believe the condition in them was due to an imperfect development of the neuroblasts of the embryonic rudiment, in consequence of which the normal restraint upon the growth of the neuroglia cells was so diminished that they underwent abnormal proliferation, and in none of these cases did the neurogliosis appear to have caused degeneration of nervous tissue. Moreover, against this theory of primary sclerosis, there does not appear to be any evidence that proliferation of neuroglia can so act. At any rate,

even in disseminated sclerosis we frequently find the nerve *cells* in a perfectly healthy condition even in the middle of a dense patch of sclerosis, and Mott in his Croonian Lectures for 1900 showed a very beautiful specimen of the spinal cord in which a bundle of nerve fibres coming from an uncut root remained perfectly healthy although surrounded by sclerotic tissue, resulting from the cutting of several adjacent nerve roots. Moreover, two of my cases of disseminated sclerosis demonstrated the same chronic degeneration in the fibres of the peripheral nerves, a situation in which there is no neuroglia, and it is a pertinent fact that by far the greater number of the islets have their origin in, and are confined to the white matter, a tissue which contains far less neuroglia than does the grey. It is also important evidence against this view of primary sclerosis that in an early and acute case of the disease (like Case No. 3) we have extensive myelin degeneration with but little sclerosis, and that careful examination of even older cases often shows that the morbid process is extending peripherally not by proliferation of neuroglia but by swelling and alteration of myelin sheaths. Occasionally, it is true, one does see some slight proliferation beyond the confines of the actual islet; but this is rare without the myelin sheaths being at the same time affected; a similar hyperplasia is sometimes seen in tract diseases, as was first pointed out by Charcot. It is of interest to remember that at one time *all* the sclerosis of the cord were considered to be due to a primary proliferation of neuroglia; and although we now know that in the systemic diseases such a view is incorrect, this theory with regard to disseminated sclerosis still finds many adherents. I think, however, in view of the considerations above advanced, that the theory is no longer tenable, and that whatever may be the cause of the nerve destruction it is certainly not due to a primary sclerosis.

Coming now to the theory of vascular obstruction, it is well known that circulatory stagnation may result from proliferative arteritis, which may, even without causing hæmorrhage, bring about localised necrosis or atrophy, and subsequent sclerosis. I have frequently observed in the spinal cords of old persons whose vessels were thickened, a diffuse and patchy overgrowth of neuroglia, presenting both to the naked eye and under the microscope an appearance somewhat like that in disseminated

sclerosis. There are nerve fibres undergoing degeneration and even naked axis cylinders, the two processes of nerve destruction and glia hypertrophy seeming to run simultaneously. As a rule, however, such areas are much more diffuse and irregular and have not the "punched-out" look characteristic of disseminated. Moreover, in these cases the vascular changes are very pronounced and readily seen, the media and adventitia often being enormously thickened and the latter sending out thickened processes radiating in all directions. But these conditions are totally distinct from disseminated sclerosis; they are confined to old people, and symptoms are either absent or are simply those of a mild paraplegia. I think the change is a senile one like that mentioned by Gull and Sutton; although a similar state of affairs may probably occur at any age where arterio-capillary fibrosis is present.

In syphilis also, the endarteritis obliterans may cause patches of necrosis which are in time replaced by islets of sclerotic tissue very much like those in disseminated. But in these cases not only are the diseased vessels characteristic, but the membranes are also usually involved, and as a rule the islets are larger in size and fewer in number. It is important to note that in these syphilitic cases the clinical resemblance may be very great, and one case which I examined and which was undoubtedly syphilitic had been during life diagnosed by a neurologist of high repute as an instance of disseminated.

I think, however, that the strongest argument against the application of this theory of vascular obstruction to disseminated sclerosis lies in the undoubted fact that numerous competent observers have utterly failed to find any evidence of such changes in this disease. Thus in the cases examined by Taylor, Huber, Sander, Buchwald, Jolly, and Frohman, the vessels were normal. Redlich noticed changes, but came to the conclusion that they could only be considered as secondary or concomitant and not the cause of the disease. Strümpell, also Schuster and Bielschowsky, are of the same opinion; and Sander finds that it is only in the oldest patches that the vessel walls are thickened and that even there they are frequently normal. In my own cases the only discoverable change consisted in some slight thickening of a few of the vessels of Cases Nos. 1 and 2, whilst in the

acute case No. 3 the vessels were perfectly normal. It is, therefore, my opinion that the slight degree of change present in these cases was simply due to the irritative action of degenerative products, a condition which may result in any case where nervous tissue has been undergoing degeneration sufficiently long, and I think it is most probable that the vascular changes which have been noticed by Rindfleisch, Charcot, Ribbert, Buss, Popoff, Bastian, Hess, Marie, and Williamson, are really secondary and not causal.

From my own cases I think it is clear that of the constituent tissues of the nervous system, the myelin substance is the first to suffer change, and that *the essential process is one of myelin degeneration independent of vascular disease*. In some cases this process may be extremely slow, and many years elapse before the disease proves fatal; in others the condition may be so rapid as practically to amount to an acute myelitis with its concomitant symptoms, and to cause death. I have already described the consecutive changes which take place in the myelin substance; at first they are of a bio-chemical nature, and it may be that at this early stage arrest of the process and complete recovery of the patient is possible, a result which is suggested by the clinical course of some cases. But once fatty degeneration has occurred such a favourable result would seem to be most unlikely; and although Charcot thought that new sheaths might be formed to replace those destroyed, I know of no evidence that any such reparation occurs; there was certainly none in my own cases.

Sooner or later the axis cylinder also undergoes degeneration, but whether this is due to it being actually attacked by the same morbid process, or whether the cylinder is incapable of independent existence after its investing sheath has been removed, I do not know. That it is eventually destroyed is, I think, certain, although in some cases it may undoubtedly persist in a naked state for some considerable time; but even then it frequently shows indication of pathological change. It may be remarked that Erben has also noticed swelling of the cylinders similar to that described in this paper. Schuster and Bielschowsky also found many of them irregularly swollen, whilst others were thin and atrophied. Sander did not observe any alteration of those cylinders within recent patches, but he states that in the older islets they had frequently disappeared. It was at one time

contended by Erben and Popoff that regeneration of the cylinder took place, until Weigert showed that what were thought to be regenerated cylinders were in reality fine neuroglia fibres.

I think that in acute cases destruction of the myelin substance within a small area is usually fairly complete before any hyperplasia of neuroglia takes place, although in the more chronic cases the two processes may be more nearly simultaneous. Hypertrophy of neuroglia fibrillæ precedes hyperplasia. The neuroglia fibres originally present first became thickened and granular looking; there is then an increased development of cells and fibres, and finally the cells become much less noticeable whilst the fibres remain as a dense network. Consequently the density and consistence of any particular islet is an indication of its age, and it is in consonance with this view that the spinal cord of Case No. 1 was so riddled with hard islets that a considerable amount of contraction and distortion of the entire cord had been produced. On the other hand, in Case 2 there occurred more islets of the intermediate character, whilst in Case 3, where degeneration was in full swing, there was very little sclerosis at all, the whole cord being swollen and distended. It must be remarked, however, that although it is most convenient to describe these islets as hard, soft and intermediate, it often happens that the three stages are present in one focus of disease; in such a case we find the most dense sclerosis towards the centre; this is surrounded by a zone of myelin degeneration with but little neuroglial overgrowth (intermediate), and this it may be by a zone characterised by perfectly normal glia but a large number of swollen medullary sheaths. Well-defined islets of softening are usually, if not always, only seen in the brain, and it would appear that in this situation either destruction and absorption of nervous matter is much more rapid or neuroglial hyperplasia more tardy.

It is clear that the medullary sheaths are affected long before the nerve cells, and the fact that in the latter the alteration is always a chronic atrophic one points, I think, to the conclusion that it is probably a secondary change resulting from the destruction of the axis cylinder processes and closely akin to the backward atrophy of cells which occurs in amputation of limbs or section of nerve roots.

I fully agree with Taylor that pigmentation is a very early and



noticeable feature in these cells, and indeed its presence in considerable amount seems to be significant of atrophic degeneration, in which repair no longer keeps pace with waste. It is true that a similar deposit is commonly seen in the ganglion cells of old persons who have not suffered from any nervous disease, then probably being a senile change; but considering the age of most of these cases, such an unusual amount of pigment must be considered as abnormal and pathological.

It would naturally be expected that pronounced paralytic and sensory symptoms should result from such extensive neuronie destruction; it is nevertheless usually stated that such are not prominent. Of many early cases, and particularly those seen in hospitals, this statement is true; but in the later stages it is undeniable that such do occur, and are a very distressing feature of the disease. I have myself seen both in infirmaries and in private practice several patients suffering from extreme paralysis with contractures, incontinence of fæces, and marked sensory disturbances, and I believe that these symptoms are quite a common feature of the later stages of this disease. There are, moreover, at least two clinical types ("spastic paraplegic" and "combined sclerosis"), in which paralytic or sensory symptoms are present from the very beginning, indeed it is likely that the majority of the cases of so-called spastic paraplegia are in reality disseminated sclerosis. The alteration in the condition of the reflexes in these cases, from exaggeration to diminution or absence, is also of considerable interest, and indeed not without diagnostic value, as indicating the spread of the morbid process from the lateral columns to the grey matter or nerve roots.

Admitting then, as I think we must admit, that the condition is primarily one of parenchymatous degeneration, we have to enquire what is the real *fons et origo mali*. We must either assume a special proneness to degeneration on the part of the myelin, or the presence of a "toxin" having a selective action upon this substance. Now, although I do not think that vascular obstructions play any part, it is nevertheless true that the distribution of the foci, both within the cerebral and spinal portions of the nervous system, is strongly suggestive of a vascular relationship, and hence gives considerable colour to the hypothesis that the various changes are initiated by a circulating toxin. At present, however, no micro-organism or toxic body has been

demonstrated, and the cholin which has been obtained from the blood of these patients is simply due to the degeneration of nerve matter. Nevertheless, this view, though purely conjectural, is highly probable, since we already know many poisons which are capable of exercising an injurious and destructive action upon nerve matter. It is at this point that chemical pathology must take the place of microscopical anatomy.

In this connection etiological data afford us but little help, since the "causes" to which the disease is attributed are of the most varied nature, and include such factors as heredity, acute infectious diseases, chronic intoxications, trauma, cold and chill, shock, fright and other mental emotions, sexual excesses, alcohol and the like. That such varying influences may actually *produce* the disease is, to say the least, extremely unlikely, although it is possible that their action as a predisposing cause may not be without effect. It may be remarked that the influence of the infectious diseases has been particularly insisted upon, although other writers, including Strümpell and Krafft-Ebing, deny the connection. With regard to the sex most affected, opinions also differ, so much so that I have thought it worth while to collect all the recently reported cases with the result that, including my own, I find there are 200 *males* as against 173 *females*.

On the whole, I think we must conclude that whilst in a considerable number of the cases one or other of these predisposing factors is present, they are not the cause of, nor indeed necessary for the production of the disease. When neuropathic conditions are present in the ancestors, a tendency to myelinic degeneration may be actually inherited; or the same tendency may be acquired in the individual in consequence of various states which cause increased stress or impaired resistance (*pathological*, e.g. infectious disease, chronic intoxications, etc.; or *physiological*, e.g. puberty, pregnancy, etc.); but the factor which actually *determines* the disease is in all probability some circulating toxin. As to the nature of this toxin we know nothing; it may be introduced from without or generated within; its very existence, indeed, is as yet hypothetical.

#### SUMMARY.

The chief points regarding the *Pathogenesis of Disseminated Sclerosis* may be summarised as follows:—

1. *The initial change is in the myelin substance; it begins as cloudy swelling and proceeds to fatty degeneration, disruption, and absorption of the myelin sheath. This process may occur in all parts of the central nervous system, also in the peripheral nerves, and a focus once started has a tendency to spread centrifugally. The existence of this degeneration can be demonstrated clinically by the presence of cholin crystals in the blood.*

2. *The axis cylinder persists for a time after the disappearance of its sheath; but later it also undergoes degenerative changes consisting of swelling and varicosity, or perhaps occasionally simple atrophy, and finally it too disappears.*

3. *As a result of this destruction of the higher elements a proliferation of neuroglia takes place, producing the patches of sclerosis. The islets occur in varying stages of consistence from soft and gelatinous to dense and firm, according to their age and the amount of neuroglia present.*

This sclerosis is therefore truly secondary in nature, and the old idea of the disease being due to a primary overgrowth of neuroglia is not supported by more recent research.

4. *As a result of the complete severance of the nerve fibre secondary degeneration takes place; if the case can be examined early enough, such is revealed by Marchi's stain; in the later stages it has been replaced by interstitial sclerosis, and the ordinary difficulties of demonstrating this are increased by the presence of the indiscriminate islets. These changes give rise to pronounced motor and sensory symptoms in the later stages of the disease.*

5. *The ganglion cells throughout the nervous system show an absence of acute change; they nearly always contain a large amount of granular pigment, and in the later stages of the disease the cells undergo a chronic atrophy which is most likely secondary to the destruction of their axis cylinder processes.*

6. *The vascular changes which occur in a certain proportion of cases must be looked upon as secondary or concomitant, and not as the cause of the nervous degeneration.*

7. *The pathological conditions are suggestive of the presence of a circulating "toxine" as the cause of the disease, but there is at present no proof that such is the case.*

8. *A predisposition to the disease may be (rarely) inherited, or*

(more commonly) induced by some antecedent pathological condition or physiological state.

9. It is important to bear in mind that some cases of multiple foci of sclerosis (following softening, the result of syphilitic vascular change) may bear an exceedingly close clinical resemblance to true disseminated sclerosis, but the two conditions are quite different in their microscopical characters.

The greater portion of the histological work in connection with these cases has been carried out in the laboratory of the London County Asylums at Claybury, to the director of which, Dr F. W. Mott, F.R.S., I wish to express my sincere thanks. The excellent micro-photographs have been taken for me by his laboratory assistant, Mr C. Geary.

#### DESCRIPTION OF COLOURED PLATES 14 AND 15.

FIG. 1.—*Teased Sections of Great Sciatic Nerve* (Marchi and Ströbe stain). (a) Normal fibre. (b) Fibre of which the myelin sheath is undergoing granular swelling. (c) Fibres showing more advanced stage of granular swelling of the sheath and early varicosity of the axis cylinder.

FIG. 2.—*Transverse Sections of Fibres from Cord* (Ströbe stain). (a) Normal fibres. (b) Fibres from the periphery of an islet in a condition of cloudy swelling. (c) Fibres from an islet showing granular swelling of myelin sheath and angular axis cylinders.

FIG. 3.—*The edge of a Sclerotic Islet in the cord* (longitudinal section, Marchi and Ströbe stain). (a) Naked axis cylinders, which cease to stain bright blue on coming within the zone of disease. (b) Loose network composed of slightly thickened neuroglia fibrils with a few naked axis cylinders which do not stain bright blue. (c) Corpuscles of Glüge containing fatty debris. (d) Droplets of myelin and fat.

FIG. 4.—*The Central portion of a Sclerotic Islet in the cord* (longitudinal section, Marchi and Ströbe stain). Showing a dense interlacement of fibrils, chiefly neuroglia; and an almost complete disappearance of products of degeneration.

FIG. 5.—*Longitudinal Section of Cord* (Marchi stain). Showing various stages of degeneration of the myelin sheath and swelling and varicosity of the axis cylinders, also free droplets of fat.

FIG. 6.—*Anterior Horn Cells from Lumbar Region*; showing early pigmentary change. (a) Cells stained by Marchi's method in which the pigment appears black. (b) Cells stained by Nissl's method in which the pigment is yellow.

FIG. 7.—*Cell of Posterior Spinal Ganglion* (Nissl stain). Showing breaking up of the chromatoplasm and deposit of yellow pigment similar to what occurs in the anterior horn cells.

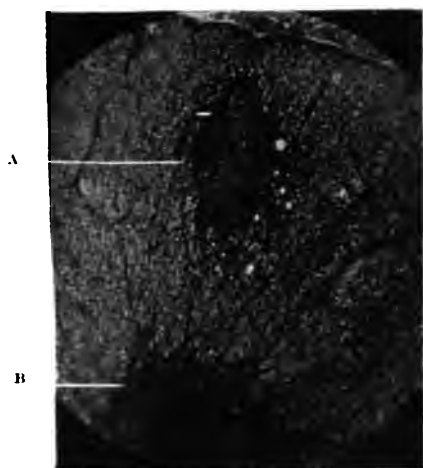


FIG. 5. Horizontal Section of Spinal Cord. A, Small dense islet of Sclerosis, with swollen fibres at periphery. B, Anterior horn of grey matter. Stained by Strøbe's method.  $\times 200$ . [E. J. S., Case No. 1.]

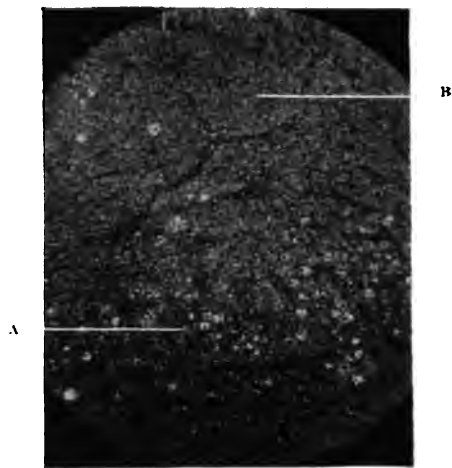


FIG. 6. Horizontal Section of Cord. A, Edge of patch of Sclerosis, showing swelling of medullary sheaths and axis cylinders. B, Normal nerve fibres. Stained by Strøbe's method.  $\times 200$ . [E. J. S., Case No. 1.]

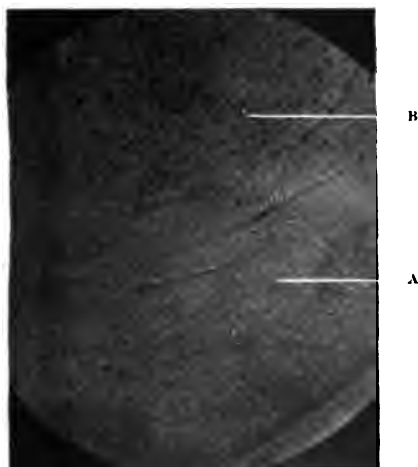


FIG. 7. Horizontal Section of Cord. A, Open patch of Sclerosis at periphery of cord, from which all nerve fibres have disappeared. B, Edge of Sclerosis with several degenerated nerves and leucocytes containing fat. Stained with Marchi's fluid.  $\times 200$ . [E. J. S., Case No. 1.]

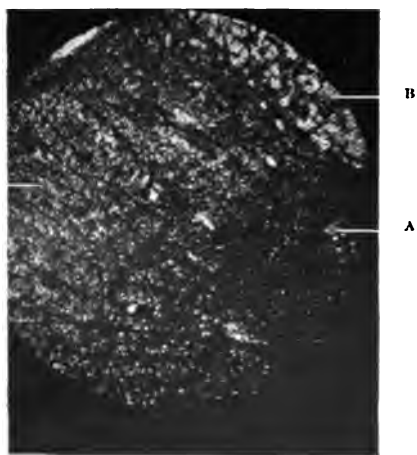
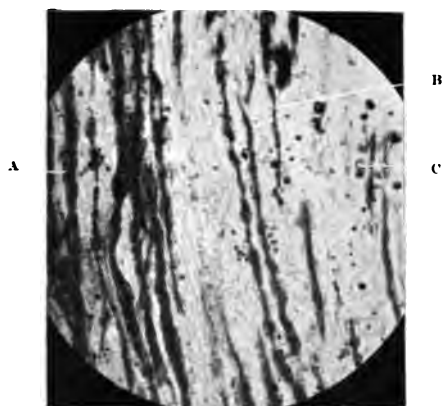


FIG. 8. Horizontal Section of Cord. A, Edge of dense patch of Sclerosis. B, Many swollen nerve fibres. C, More open Sclerosis at periphery of cord from which nearly all the nerves have disappeared. Stained by Strøbe's method.  $\times 200$ . [E. J. S., Case No. 1.]





B

C

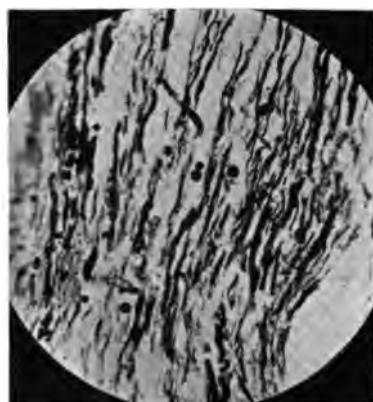
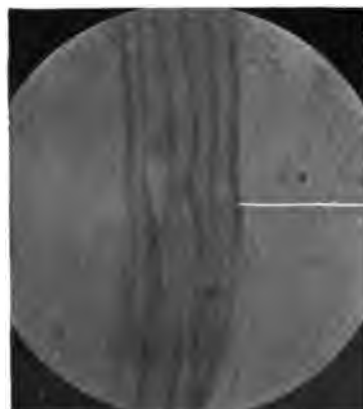
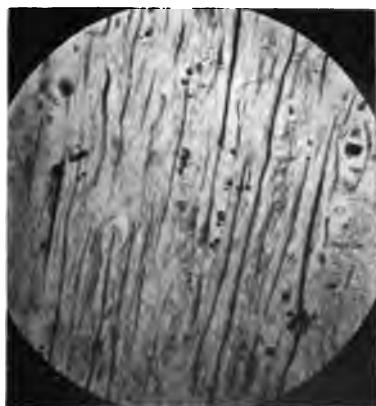


FIG. 9. Longitudinal section of the cord near the edge of a sclerotic patch. A, Fibres showing swollen condition of sheath. B, Sheath becoming detached. C, Droplets of fat and myelin resulting from degenerated sheaths. [J. S., Case No. 2, Marchi-Pal.  $\times 330$ .]

FIG. 10. Longitudinal section of cord, showing acute changes in the myelin substance with free droplets of fat and myelin. [H. B. R., Case No. 3, Marchi-Pal.  $\times 330$ .]



A

FIG. 11. Longitudinal section of cord showing a sclerotic islet containing nerve fibres, droplets of myelin and fat, and fine interlacing neuroglia fibres. [H. B. R., Case No. 3, Marchi-Ströbe.  $\times 330$ .]

FIG. 12. Longitudinal section of great sciatic nerve, showing at A, fibres undergoing degeneration. [J. S., Case No. 2, Paraffin section, Marchi.]





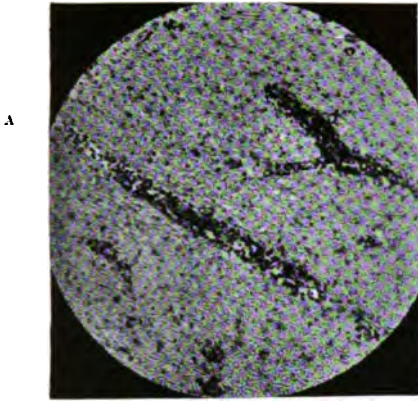


FIG. 13. Horizontal Section of Cord showing open patch of Sclerosis from which most of the nerve fibres have disappeared. Greatly distended vessels are seen crossing the patch (A), the lymphatic sheaths of which are much dilated and filled with leucocytes. [E. J. S., Case No. 1. Stained logwood and eosin.  $\times 200$ .]

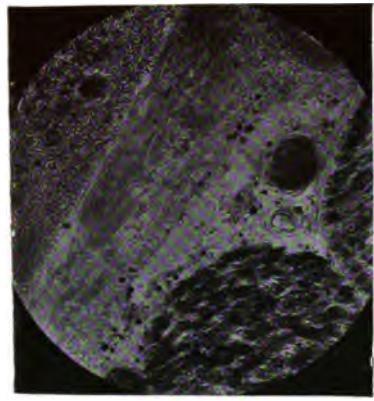


FIG. 14. Horizontal Section of Cord. A, Periphery of cord. B, Nerves of posterior root. Between these are seen greatly distended vessels, and in the lymphatic spaces many red blood corpuscles and leucocytes filled with fat. [E. J. S., Case No. 1. Stained with Marchi's fluid.  $\times 330$ .]

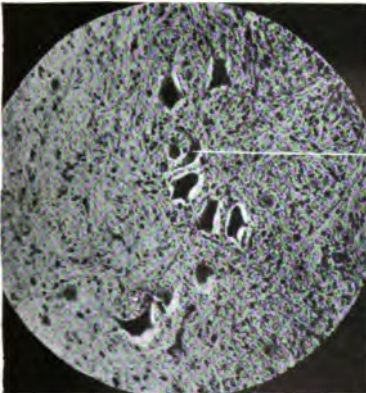


FIG. 15. Group of cells of anterior horn, showing scattered amongst the normal ones several (A) in a condition of degeneration (shrunken, outline irregular, staining differently). Stained by Ströbe.  $\times 200$ . [E. J. S., Case No. 1.]

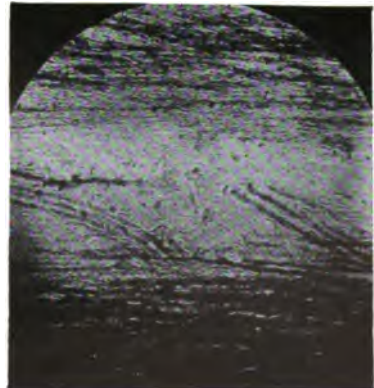
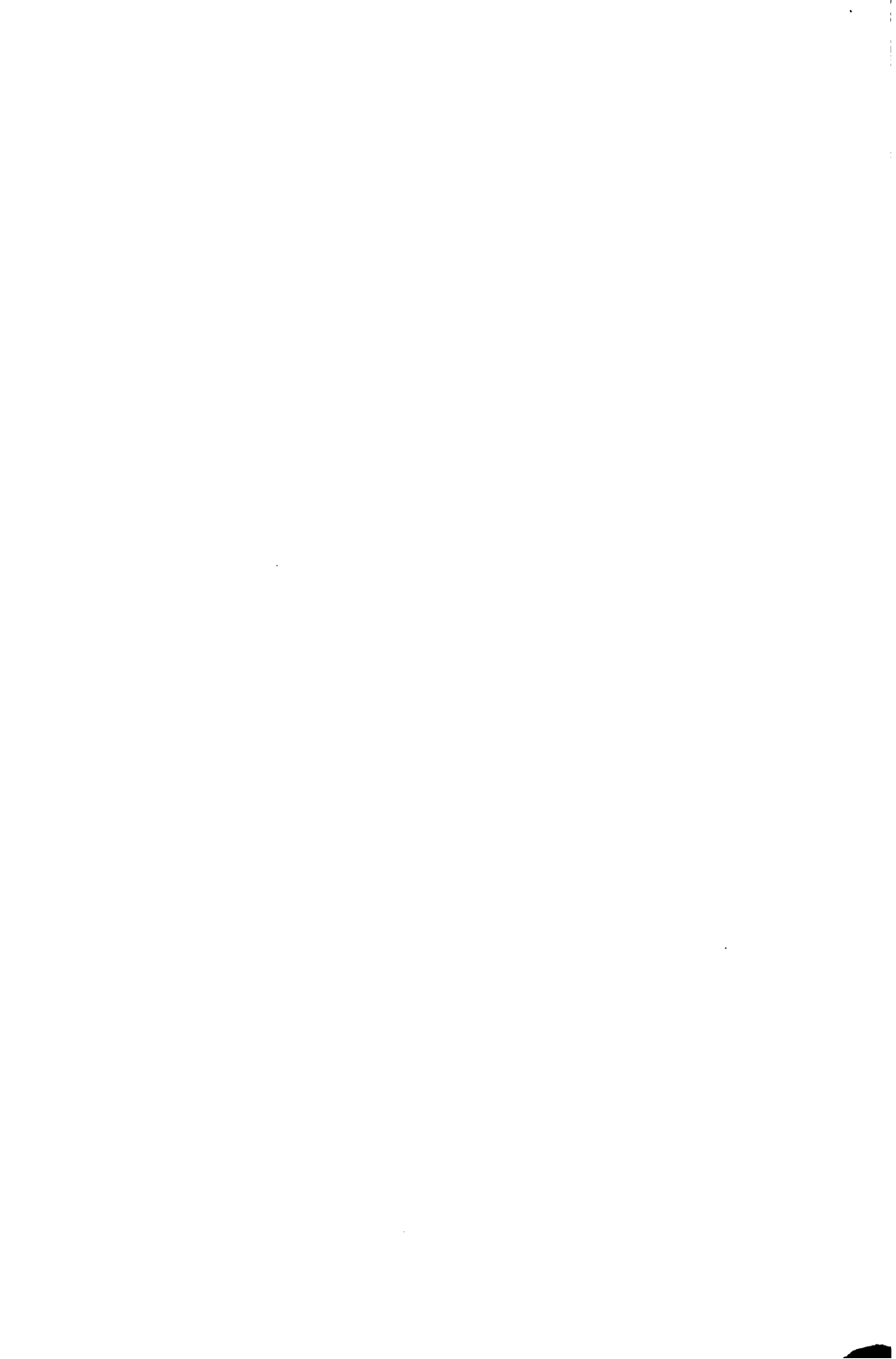


FIG. 16. A, Longitudinal section of an islet of Sclerosis with a few normal fibres crossing obliquely—most of the fibres having disappeared. B, Normal cord. [E. J. S., Case No. 1. Stained by Weigert-Pal and eosin.  $\times 200$ .]





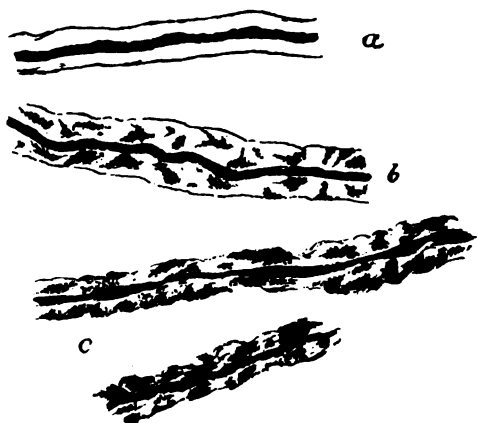


FIG. 1.

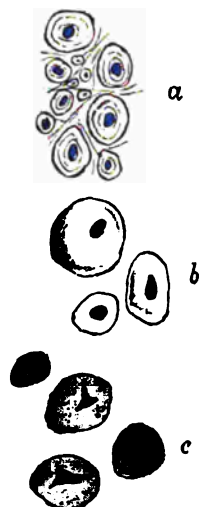


FIG. 2.

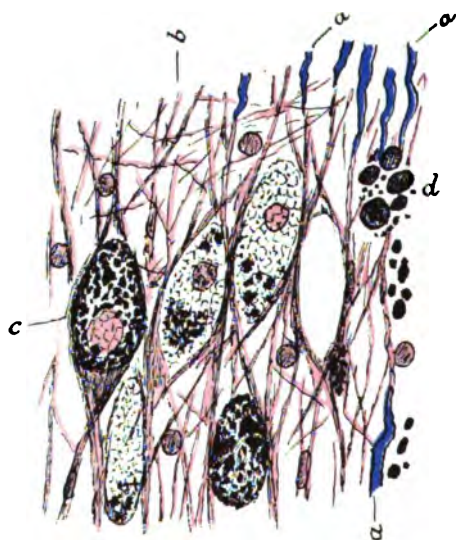


FIG. 3.



FIG. 4.

DISSEMINATED SCLEROSIS.

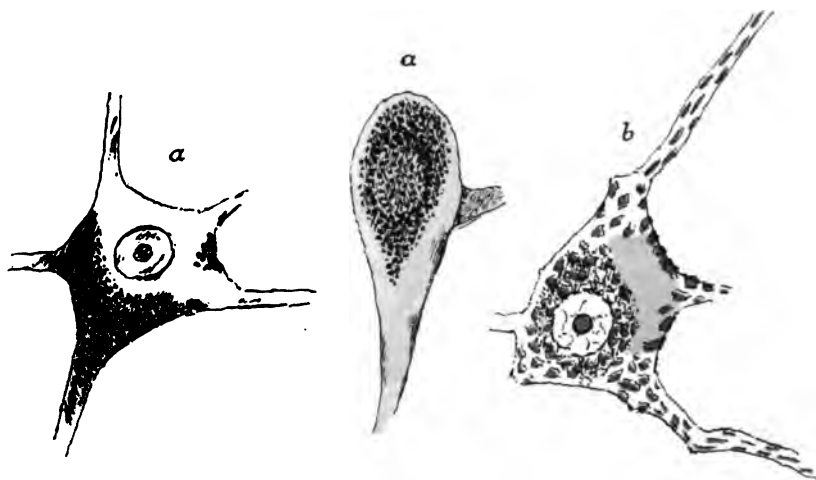


FIG. 6.



FIG. 5.

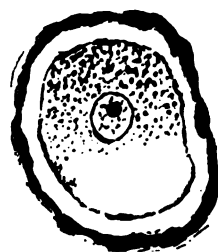


FIG. 7.

*A. F. Fredgold.*  
del.  
1908.

DISSEMINATED SCLEROSIS.



## REFERENCES.

- Adamkiewicz, "Die degenerativen Erkrankungen des Rückenmarks," Stuttgart, 1888.
- Babinski, *Archives de Physiol.*, vol. v., 1885.
- Bourneville and Guérard, "De la Sclérose en Plaques Disséminées," Paris, 1869.
- Buchwald, *Deutsch. Arch. für klin. Med.*, vol. ix., 1872.
- Blumreich and Jacoby, *Neurol. Centralbl.*, 1898.
- Von Bechterew, *Neurol. Centralbl.*, 1896 and 1899.
- Bruno Leick, *Neurol. Centralbl.*, 1899.
- Buss, *Deutsch. Arch. für klin. Med.*, Band xiv.
- Bruns, *Berlin. klin. Woch.*, vol. xxv., 1888.
- Charcot, "Diseases of the Nervous System," New Sydenham Society.
- Déjérine, *Revue de Médecine*, 1884.
- Demange, *Revue de Médecine*, 1885.
- Eichhorst, *Virchow's Archives*, 1896.
- Erben, *Neurol. Centralbl.*, 1898.
- Freund, *Archiv. f. Psychiatrie*, vol. xxviii., 1890.
- Fürstner, *Neurol. Centralbl.*, 1895.
- Gowers, "Diseases of the Nervous System," 2nd Ed., vol. ii., 1893.
- Goldscheider, *Zeitschrift f. klin. Med.*, vol. xxx., 1896.
- Gutzler, *Neurol. Centralbl.*, 1896.
- Henschen, *Neurol. Centralbl.*, 1899.
- Hess, *Archiv. f. Psychiatrie*, vol. xix., 1888.
- Huber, *Virchow's Archives*, vol. cxi., 1895.
- Jacobson, *Neurol. Centralbl.*, 1895.
- Jolly, *Archiv. f. Psychiatrie*, vol. iii., 1872.
- Jordan, *Birmingham Med. Review*, vol. xxxi., 1892.
- Kaiser, *Neurol. Centralbl.*, 1889.
- Koppen, *Archiv. f. Psychiatrie*, vol. xvii., 1886.
- Krafft-Ebing, *Neurol. Centralbl.*, 1896.
- Krafft-Ebing, *Neurol. Centralbl.*, 1899.
- Kahler and Pick, *Prager Vierteljahrschrift*, 1897.
- Koenig, *Berlin. klin. Woch.*, No. 39, 1895.
- Leyden, *Zeitschrift f. klin. Med.*, vol. xxi., 1892.
- Leyden-Goldscheider, "Nothnagel's Pathology," vol. x., 1897.
- Lapinsky, *Zeitschrift für klin. Med.*, vol. xxviii., 1895.
- Marie, P., "Diseases of the Spinal Cord," New Sydenham Society.
- Mendel, *Neurol. Centralbl.*, 1897.
- Mendel, *Deutsch. med. Woch.*, vol. xxiii., 1897.
- Nolda, *Archiv. für Psychiatrie*, vol. xxiii., 1892.
- Nonne, *Neurol. Centralbl.*, 1898.
- Oppenheim, *Neurol. Centralbl.*, vol. xv., 1896.
- Orlowsky, *Neurol. Centralbl.*, 1897.
- Obersteiner, "Nervöse Central. Organe," etc., 3rd Ed., 1896.
- Popoff, *Neurol. Centralbl.*, vol. xiii., 1894.
- Probst, *Deutsch. Zeit. f. Nervenheilkunde*, vol. xii., 1896.
- Ribbert, *Virchow's Archives*, vol. xc., 1882.

- Rossolimo, *Neurol. Centralbl.*, 1896.  
 Redlich, *Neurol. Centralbl.*, 1896.  
 Rindfleisch, *Virchow's Archives*, vol. xxvi., 1863.  
 Russell, Risien, "Clifford Allbutt's System of Medicine," vol. vii  
 Schultze, *Neurol. Centralbl.*, vol. iii., 1884.  
 Schuster and Bielschowsky, *Neurol. Centralbl.*, vol. xvi., 1897.  
 Schuster and Bielschowsky, *Neurol. Centralbl.*, 1899.  
 Sander, *Neurol. Centralbl.*, 1898.  
 Sander, *Neurol. Centralbl.*, 1899.  
 Strümpell, *Archiv. f. Psychiatrie*, vol. x. No. 3.  
 Sachs, *Journal of Nervous Diseases*, 1898.  
 Taylor, E. W., *Deutsche Zeit. f. Nervenheilkunde*, vol. v., 1894.  
 Totzke, *Dissert. Berlin*. 1893.  
 Torti and Angelini, *Neurol. Centralbl.*, No. 24, 1893.  
 Uhthoff, *Archiv. für Psychiat.*, vol. xxi., 1891.  
 Weigert, "Beiträge zur Kenntniss der normalen menschlichen Neuroglia,"  
 F. M., 1895.  
 Williamson, *Medical Chronicle*, March 1894.  
 Westphal, *Archiv. für Psychiat.*, vols. ix. and xiv., 1883.

## A METHOD OF PRODUCING A SELECTIVE DEPOSIT OF SILVER IN BLOCKS OF TISSUE AND OF OBTAINING SECTIONS SUITABLE FOR TONING WITH GOLD, PLATINUM AND OTHER METALS.

By W. FORD ROBERTSON, M.D.

ALTHOUGH silver salts have long been employed in histological work, no one has yet succeeded in devising a satisfactory method, capable of general application, by which a selective deposit of the metal may be produced in blocks of tissue. No such process can be regarded as satisfactory if it entails injury to the structural elements, the formation of disfiguring precipitates, or excessive deposit in a superficial zone. It is further requisite that the degree of the silver impregnation should be under control, and that it should be possible to obtain sections upon which toning processes, similar to those used for photographic silver prints, can be carried out successfully. A method of this nature might be expected to yield histological pictures far ex-



ceeding in sharpness of outline and wealth of detail those that can be obtained by any dyeing process at present in use, and the preparations would certainly be quite permanent.

The nearest approach we as yet have to the attainment of a process of this kind is represented in the recently described methods of Bielschowsky<sup>1</sup> and Ramon y Cajal.<sup>2</sup> Bielschowsky's process consists essentially in subjecting tissues, while charged with formalin, the reagent in which they have been fixed and hardened, to the action of a strong solution of ammonio-nitrate of silver, and in subsequently effecting the further reduction of the silver compounds present by means of alkaline formalin. In the recent modification of his method, applicable only to sections, silver nitrate is employed, but the chemical changes involved are virtually the same. Whilst Bielschowsky's methods are unquestionably of great value for the demonstration of the neurofibrils, they can hardly be regarded as furnishing a solution of the problem of how to attain a satisfactory process of impregnating tissues with silver. They involve the formation of precipitates only soluble in reagents which would also dissolve the selective silver deposit in the tissue-elements, and when they are applied to blocks of tissue the impregnation is not uniform, amounting to an incrustation near the surface and tending to be deficient in the centre.

Ramon y Cajal's recent methods, though probably still more valuable for the demonstration of the neurofibrils, and though supplying us with a ready means of studying the clubs or buttons of Auerbach, come no nearer the fulfilment of the requirements of a satisfactory general process for silvering tissues. The application of a solution of silver nitrate to fresh tissues results in serious injury to them in a histological sense, and the more recent modifications in which there is a preliminary hardening either in alcohol or in formalin serve only for the demonstration of special tissue-elements.

In 1898, I endeavoured to work out a histological process in which advantage was taken of the powerful reducing action of formaldehyde upon ammonio-nitrate of silver, as in the methods of Bielschowsky. Blocks of formalin-hardened tissues

<sup>1</sup> *Neurologisches Centralbl.*, 1902, n. 13; *Ibid.*, 1903, n. 21.

<sup>2</sup> *Archives latines de Médecine et de Biologie*, t. 1, n. 1, 1903; *Bull. de la Soc. de Biologie*, 4 mars 1904. See *Rev. of Neurology and Psychiatry*, April 1904, p. 293.

were exclusively used. The results obtained were unsatisfactory and the experiments were abandoned. The defects which it was found impossible to remedy were especially unevenness of the impregnation, the formation of precipitates and seeming unsuitability of the sections for toning with gold. Preparations were, however, obtained, in which some parts gave very clear pictures of the neurofibrils. Some months ago I began a series of experiments with the purpose of endeavouring to perfect this histological process, and have now succeeded in overcoming the difficulties previously experienced.

It was ascertained that any serious unevenness of impregnation could be prevented by washing out excess of formalin from the tissues before immersion of the blocks in the silver bath. It was further found that the formation of precipitates could be obviated by allowing the reduction to be effected simply by the formaldehyde or its derivatives remaining fixed in the tissues, and by completely washing out the silver salts with ammonia and water before toning. The use of a special reducing bath was found to be not only superfluous, but in certain respects detrimental. Lastly, it was ascertained that sections of tissues so prepared were capable, after neutralisation of the ammonia, of being satisfactorily toned by what are essentially the ordinary photographic processes. Experience has, however, shown that an acid toning bath, used in a particular way, is specially suitable.

Whilst the general principle of the method is adhered to, the process, it will be evident, admits of great variation in its details, with corresponding modification of the result obtained. Instead of ammonio-nitrate of silver, other combinations of a silver salt and ammonia may be used. A solution prepared by saturating  $\frac{1}{2}$  per cent. ammonia in water with silver carbonate, and a similar solution of silver acetate, have been especially successful. The result may also be influenced by varying the strength of the ammonio-silver solution employed and by adding an excess of ammonia. The extent to which the formalin has been washed out of the block also materially affects the character of the silver impregnation. Another important factor is the length of time the tissues are allowed to remain in the silver bath, the impregnation generally being slowly progressive for a considerable period. Lastly, by the employment of various toning processes,

such as are used in photographic work, many modifications of the final result may be obtained in a single lot of sections.

Whilst the method is thus capable of being modified in its details in innumerable ways, it will conduce to clearness if the exact process that has so far been found most generally serviceable is fully described in its various stages. It is as follows :—

1. Wash thin slices of formalin-hardened tissues (5 to 10 per cent. solution of formalin in water ; change on second day) for about twenty-four hours in a bowl of tap water. Change the water at least once.

2. Transfer to an ammonio-nitrate of silver solution, prepared by adding to a 1 per cent. solution of silver nitrate in distilled water a 5 per cent. solution of ammonia in distilled water, drop by drop, until the precipitate which forms is nearly but not entirely dissolved, and then filter. The fluid should measure at least fifty times the volume of the tissues. Cork the bottle or specimen tube and put it in the dark. The pieces are ready for examination in from one to ten weeks.

3. Place a piece of the impregnated tissue in a bowl of water (500 c.c.) to which about 3 c.c. of 5 per cent. ammonia have been added. Renew the fluid after about an hour, and leave the tissue in it for three or four hours longer.

4. Transfer to dextrine solution (dextrine 5 ounces or 140 grammes, water 10 ounces or 280 c.c. ; dissolve by boiling ; filter through cotton wool while still hot ; after it has cooled add 1 per cent. of carbolic acid) to which ammonia has been added in the proportion of 10 drops of a 5 per cent. solution to one ounce, immediately before use. Allow the tissue to remain in this for from twelve to twenty-four hours.

5. Cut thin sections on the freezing microtome. Transfer them from the knife to a bowl of water to which about 10 drops of 5 per cent. ammonia have been added. After about five minutes transfer the sections to another bowl of ammonia and water, and after a similar period give them a third wash.

6. Transfer the sections to a bowl of water to which there have been added from 5 to 10 drops of a saturated solution of citric acid in water, and allow them to remain in this for four or five minutes.

7. Place the sections in a bowl of tap water and after a few

minutes transfer them to a second bowl of water. They are now ready for toning.

8. To 10 c.c. of a  $\frac{1}{2}$  per cent. solution of gold chloride in distilled water, add a single drop of a 1 per cent. solution of citric acid in water, and filter the fluid, preferably into a flat-bottomed white porcelain dish. Transfer the sections from the water to this toning bath by means of a glass rod or platinum needle. Allow them to remain spread out for about one hour. About a dozen sections of ordinary size may be toned in this amount of the gold solution.

9. Place the sections for about half a minute in a bowl of tap water, and then transfer to a bowl of water to which 10 drops of 5 per cent. solution of ammonia have been added, where they should remain for two or three minutes.

10. Transfer to a bowl of tap water.

11. Dehydrate the sections with absolute alcohol, clear in equal parts of turpentine and benzole, remove turpentine with pure benzole, mount in benzole balsam.

In the sections prepared for toning we have a deposit of metallic silver impregnating the different constituents of the tissues in various degrees. By the prolonged action of the acid gold toning bath, the silver is probably entirely replaced by gold. The colour of the tissues is changed from various shades of yellow and brown to red and reddish black. The result is in part that of a sharp basic stain, but many acidophile tissue-elements also tend to be deeply impregnated. Among these are the neurofibrils, both in their intra-cellular and extra-cellular course. Some preparations have also been obtained in which the clubs or buttons of Auerbach are impregnated, and there is no reason to doubt that the method can be made to serve for the demonstration of these important structures. The method is, however, put forward not as one for the demonstration of any particular tissue-element, but as a process that is capable of very general application in normal and pathological histology. At the same time it is claimed that it is of special value for the study of the nervous system. Thus it has already served to show the partial disintegration of the neurofibrils in the body of many of the cortical nerve cells in general paralysis.

Platinum toning of the silvered sections may be carried out

as follows. To one ounce of freshly prepared  $\frac{1}{2}$  per cent. solution of potassium chloroplatinite in water, add ten drops of a 5 per cent. solution of phosphoric acid in water (citric acid may be used instead). Filter this solution and place the sections in it for about half an hour. Wash the sections shortly in water and then clear them in a 20 per cent. solution of nitric acid in water, in which they should remain for about five minutes. Thereafter wash the sections well in water, dehydrate, clear and mount in balsam.

If alkaline or neutral gold toning solutions (e.g. 1 per cent. solution of borax in water, 5 to 10 c.c., and 1 per cent. gold chloride solution about twenty drops) are used, they should be allowed to act generally for about half an hour. The sections should subsequently be treated as in the acid gold toning process already described.

The silvered sections, after having been carefully washed in ammonia and water, may, if desired, be preserved in 1 per cent. formalin. They must then be thoroughly washed in water before being toned. Sections that have been toned with gold or platinum, and washed as directed, may be preserved in the same way.

---

## Abstracts

### ANATOMY.

- A FEW FACTS REGARDING THE ARRANGEMENT OF THE**  
(214) **MOTOR CELLS AT THE LEVELS OF ORIGIN OF THE**  
**NERVES OF THE EXTREMITIES.** G. BIKELES, *Neurolog.*  
*Centralbl.*, May 1, 1904, p. 386.

THIS paper is only a short note on the arrangement of the motor cells of the anterior cornua in the cross section of the spinal cord at the level of origin of the nerves to the extremities.

His results are:—

- (1) The ventro-medial group supplies the musculature of the vertebral column and the muscles innervated by the rami dorsales.

- (2) The call groups for the proximal muscles of the limbs are placed in each segment ventral to those for the distal muscles.
- (3) The motor cells for the dorsal muscles of a myotome constantly lie lateral to those of the ventral muscles in the cross section of the anterior cornua.
- (4) It is this arrangement of the motor cells as they correspond to the dorsal or ventral, distal or proximal muscles of the limbs which gives the appearance of nervous localisation in the spinal nerves.

He draws the conclusion that as the arrangement of motor cells according to constant morphological laws is the best guarantee for their exact function, it is needless to postulate an anatomical representation of physiological functions, as physiological factors are sufficient for their harmonious correlation.

GORDON HOLMES.

**INNERVATION OF THE MAMMALIAN EYE.** Preliminary Communication. MAX BIELSCHOWSKY and BERNHARD POLLACK, *Neurolog. Centralbl.*, May 1904, p. 387.

BIELSCHOWSKY has already described in this *Centralblatt* (1903, No. 21) a method for impregnating the central nerve organs with silver, which shows the intracellular fibrillæ, Golgi network, and axis-cylinders. In the original method the impregnation is made on frozen sections. Since the technique when applied to the eye, especially the retina, offers great difficulty, it has been altered in a way that allows of paraffin embedding. The results recorded in this communication were obtained chiefly by the following method:—

1. The material, as fresh as possible, is fixed in 12 per cent. formol. The globe is previously opened behind the ciliary body, and the vitreous removed. The retina is detached and further treated. Iris and cornea may or may not be separated.

2. The tissue is then placed in a 2 per cent. watery solution of silver nitrate for 24 to 48 hours.

3. After rapid immersion in distilled water the tissue is transferred to the following fluid, which must be fresh:—To 20 c.cm. of a 2 per cent. silver nitrate solution two or three drops of a 40 per cent. sodium hydrate solution are added.  $\text{Ag}_2\text{O}$  is formed. During constant stirring with a glass rod ammonia is added until the precipitate is dissolved. By this procedure two ammonium silver salts are formed,  $\text{N}(\text{NH}_4)\text{AgH}_2\text{NO}_3$  and  $\text{Ag}_2\text{O} \cdot 2\text{NH}_3$ , of which the latter especially is distinguished by its great reducing power. The objects remain in the solution from a half to one hour, or a little longer.

4. After rapid immersion in distilled water the object is placed for 12 to 24 hours in a 20 per cent. formol solution, which causes a marked reduction of the ammonium silver salt.

5. The tissue is then as quickly as possible dehydrated in alcohol of increasing strengths, and after treatment with xylol and xylol-paraffin embedded in paraffin at medium melting-point in the usual way.

6. From the blocks serial sections are made, and are fixed to the slides with glycerine-albumen.

7. For obtaining permanent preparations and producing a marked differentiation of the nerve tissue it is necessary to use gold or platinum. After preparing in the usual way for aqueous solutions (xylol, absolute alcohol, spirit), the sections are placed in a neutral, alkaline, or weak acid gold-bath. For choice, Bielschowsky added two or three drops of a 1 per cent. gold chloride solution to 10 c.cm. of water, and acidulated with two or three drops of acetic acid. Weak acid platinum baths give lasting results, but less contrast is produced.

8. To remove the insufficiently reduced silver, the sections which receive a small addition of a solution of acid sodium sulphite (a drop of the concentrated solution in 10 c.c. of water) in the acid gold-bath are placed in a 5 per cent. solution of sodium hyposulphite ( $\text{Na}_2\text{S}_2\text{O}_3$ ) for not longer than half a minute. After careful washing in distilled water they are dehydrated, cleared in carbol-xylol, and mounted in Canada balsam. Besides impregnation in block and subsequent embedding in paraffin, impregnation of frozen sections sometimes gives good results. Lastly, the impregnated piece after procedure 4 and long immersion in water may be frozen and cut.

The investigations were carried out on eyes of pigmented and non-pigmented rabbits, horses, dogs, and man. Many of the results obtained by the writers are of value only in that they confirm previous descriptions for the most part based on the gold method. The paper is intended more to indicate the scope of the method than to draw attention to what is new or little known.

C. H. USHER.

**ON THE EXTERNAL APPEARANCE OF THE DENDRITES OF  
(216) THE NERVE CELLS OF THE ANTERIOR AND POSTERIOR  
CORPORA QUADRIGEMINA OF HIGHER VERTEBRATES  
(RABBIT AND MOUSE). CZARNIECKI, *Nouv. Icon. de la  
Salpêtrière*, mars-avril 1904, p. 100.**

The rabbits and mice used for the research were killed by decapitation, and the corpora quadrigemina treated by the rapid Golgi-Ramon y Cajal method.

Sections were made in a direction perpendicular to the aqueduct of Sylvius.

In the peripheral part of the anterior quadrigeminal body the dendrites pursue an irregular course towards the surface. They ramify, become thinner at their distal extremity, and there the gemmulæ are more numerous.

The gemmulæ may be straight or curved, ending in a spherical globule, and in some instances two or three may arise from a common stalk. On the dendrites there often occur varicosities or moniliform swellings.

In the ventral part of the anterior quadrigeminal body the dendrites ramify little and show few gemmulæ.

In the posterior quadrigeminal body the dendrites soon after leaving the cell give off a large number of ramifications which narrow gradually. On the basal part of the dendrites there are no gemmulæ. On the fine terminal branches there are swellings but no gemmulæ.

The cell body is almost always large and spherical, while the dendrites are fine and short.

According to the author the dendrites of the nerve cells in the anterior quadrigeminal body greatly resemble those of the cells in the spinal cord. Those of the anterior cornua resemble dendrites of cells in the ventral part of the anterior quadrigeminal body, while those in the posterior horns are similar in appearance to the dendrites of cells in the peripheral part.

DAVID ORR.

**RESEARCHES UPON THE STRUCTURE OF THE FIBRILLAR  
(217) PART OF THE NERVE CELLS IN NORMAL AND  
PATHOLOGICAL STATES. G. MARINESCO, *Rev. Neurol.*,  
No. 9, 1904, p. 405.**

MARINESCO has carried out an investigation with Ramon y Cajal's new silver method, and has obtained results which confirm the recent observations of the latter regarding the neurofibrils. He also defends the neuron theory against recent attacks, and records a number of original observations upon the appearances presented by the fibrils in normal and pathological conditions, more especially in their course within the nerve cells.

He states that the disposition of the neurofibrils in the different types of nerve cells is so variable, that it is difficult to give a general description of their morphology and of their course within the cytoplasm. He considers that their arrangement, form and course are entirely subordinate to the form and size of the nerve cell. As Cajal has pointed out, most nerve cells show a superficial and a



deep fibrillar network, the latter being the denser. They are separate at the base of the cone of origin of the axis-cylinder, but blend at the point at which the cone becomes the axone. In some cells it is easy to recognise a reticular structure. In many, however, the fibrils are so densely packed that it is impossible to say whether they anastomose or merely cross. Cells of an elongated form are of the fasciculated type, the fibrils appearing to traverse them from pole to pole without forming a network. Whilst not denying that there may be fibrils which thus pass through the cell without branching, Marinesco is inclined to believe that they commonly give off lateral ramifications. He distinguishes two kinds of fibrils, namely, those which, though they contribute branches to the intra-cellular network, do not lose their individuality, and those which break up entirely in this network.

In the cells of Purkinje the neurofibrils present a very distinct reticular structure, especially in the new-born animal. The author confirms the statement of Cajal, opposed to the opinion of Bethe, that the granules are nerve cells. They have a fibrillar network and their dendrites and axones contain neurofibrils.

Most of the pyramidal nerve cells of the cerebral cortex are of the fasciculated type. A network may be seen at the base of the pyramid. When the axone is situated laterally, fibrils often pass into it directly from the principal protoplasmic process.

In the cells of the spinal ganglia, the method gives pictures which correspond with those already obtained by the use of Delafield's hæmatoxylin, thus confirming the view that these cells contain a true reticulum.

The new method serves admirably for the study of the pericellular plexus and its terminations on the cell-wall, though certain special details of technique are necessary for their demonstration. Cajal designates these terminations the clubs or buttons of Auerbach. They appear as black, often granular bodies; sometimes the central part is comparatively clear. They are exceedingly abundant on the cell-bodies and protoplasmic prolongations of many of the cells of the cord and bulb, but they have not yet been demonstrated in those of the cerebral cortex. The method does not reveal the pericellular network of Golgi, which, it is now becoming evident, is an artificial structure.

Cajal has found that ten days after section of the hypoglossal nerve of the rabbit, the corresponding nerve cells, in preparations by his new method, show certain definite structural alterations, consisting in pallor, reddish colour and granularity of the fibrils, disappearance or narrowing of the spaces normally occupied by the Nissl bodies and pallor of the nucleolus. Marinesco has made a series of similar observations upon the cells of origin of the hypoglossal, vagus, facial and spinal motor nerves. He finds that the alterations vary according to the severity of the nerve lesion,

being most marked when the nerve has been torn away, and least after simple section. Sixty hours after tearing out of the hypoglossal, the nerve cells of the nucleus are swollen and pale; the fibrils of the cell-body are granular, and the normal reticular appearance is lost. The fibrils of the prolongations are less affected than those of the cell-body. Four days after the same nerve lesion, the cell changes are more advanced. Fragmentation of the fibres has extended to the processes. After eight days, the cells are diminished in volume and have lost most of their processes. They have a granular appearance and brownish red colour.

The nerve cell changes characteristically resulting from section of a nerve are described as seen in the nucleus of the facial. They consist in pallor, reddish coloration (in place of black), granularity and approximation of the fibrils, and more or less complete disappearance of the reticulum. He thinks that the disappearance of the network after simple section may be due to dissolution of the chromatophile substance and approximation of neurofibrils, in consequence of which the cell assumes a striated appearance. At the time when the chromatophile substance is being reformed, the neurofibrils tend to return to their normal condition. He adheres to the opinion that these neurofibrils when destroyed cannot be formed anew.

He has also observed fragmentation of the neurofibrils and other changes in the nerve cells of the spinal cord within four and a half hours after ligature of the abdominal aorta. In rabies he has found, in confirmation of an observation of Ramon y Cajal's, fusiform thickening of the neurofibrils in the cells of the cord, bulb, and spinal ganglia. He thinks, however, that this lesion is a transient one, as the majority of the cells show more profound changes, consisting in actual disintegration of the fibrils.

W. FORD ROBERTSON.

## PSYCHOLOGY.

**THE PSYCHOLOGY OF JOAN OF ARC.** JULES DUMEZ, *Ann. Méd.* (218) *Psychol.*, May-June 1904, p. 353.

NOWHERE, perhaps, can fuller evidence be obtained of divine intervention in human affairs than in the story of Joan of Arc. No wonder, then, that generation after generation, the believer and the sceptic alike find their attention drawn to the strange story of the Pucelle, at once simple village maiden and man of war. The point of view of the present writer is perhaps sufficiently indicated in his remark that nowadays the celestial as well as the infernal powers are having a little rest, since gravitation, electricity, the X-rays,

heredity, atavism, microbes, and all the biological and psycho-chemical energies have become the administrators of the facts of nature. It is impossible to do any justice to his paper in a mere summary, for his argument is based upon a very interesting *précis* of the evidence given at the trial of Joan of Arc in 1430 which ended in her condemnation, and of the depositions of witnesses in the process of rehabilitation in 1456, and this will not bear any compression. Suffice it to say that in the unconscious mind, which has had so much to answer for of late, the writer finds the explanation of the phenomena which in so extraordinary a fashion restored the fortunes of France. The hallucinations of which Joan was the subject could be interpreted by her only as due to real personages who, as she believed, had come down from heaven to counsel and direct her. But in our day the sane subject of such an experience would recognise that the perception was purely imaginary, and without reality. In Joan we recognise a double personality, in which the one self was able to converse with the other. The conscious self of the little illiterate girl argued and recoiled, knowing nothing of fighting or riding; while her other self, that of the captain, entered the sphere of consciousness in the form of hallucinations. This second self appeared strange to her, a real apparition, a celestial material being, in the alternative form of St Michael, St Catherine, St Margaret. It may happen to us that on awaking in the morning there dawns on us the solution of a problem which troubled us at night, or there may break upon us unexpectedly the answer to a question which has been worrying us for days. This implies that mental work has been going on outside our consciousness. So was it with Joan, but with the peculiarity that when the sub-conscious became conscious there were accompanying visual and auditory hallucinations, and it was to this hallucinatory consciousness that she gave the name of St Michael, St Catherine, St Margaret, according to the mental images presenting themselves. She regarded herself as receiving inspiration from God, although she was herself elaborating, sub-consciously, the counsels she received. Could she escape such an explanation? No. In her time everything in the world, nature and humanity, was explained by God or by the devil.

Does such an explanation as has been attempted detract from the merits of the Maid of Orleans? On the contrary, it is only in our day that Joan of Arc appears in all the glory of her genius. For us, she is no puppet of which God or the devil pulls the strings. She is a human being, great in her energy, her intelligence, her self-sacrifice, all devoted to the greatest, the most noble of causes, the delivery by arms of her country.

W. B. DRUMMOND.

## PATHOLOGY.

**THE EXPERIMENTAL SOLAR SYNDROMES.** LAIGNEL-LAVASTINE, (219) *Journ. de Neurol.*, April 20, 1904, p. 98.

COMPLETE removal of the solar plexus in the dog was found to be a very serious operation. Half the animals died within twenty-four hours, and many of those which survived the shock of the operation died in collapse four or five days afterwards. When an animal lived beyond the fourth or fifth day the symptoms began to pass off and as a rule it survived.

The complex of symptoms produced by removal of the solar plexus the author has termed the *solar syndrome of paralysis*. This may be very acute, acute, sub-acute or chronic. Stimulation of the plexus (by dragging on or pinching the nerve filaments) produces very severe effects, sometimes even death. These effects he calls the *solar syndrome of excitation*, and by the *solar symptoms of irradiation* he means the indirect symptoms produced by excitation of the plexus while the splanchnic and vagi nerves are intact.

The *solar syndrome of paralysis*, in its most acute form, consists in a great fall of blood pressure, rapid and feeble pulse, coldness of the extremities, vomiting, diarrhoea, anuria and general weakness which ends in death within twenty-four hours. In less acute cases there is general weakness, rapid and feeble pulse, vomiting, foetid diarrhoea, urine scanty and high coloured from the presence of bile pigments and indican. When death takes place on the fourth or fifth day there is found to be great hyperæmia of the mucous membrane of the gastro-intestinal canal, and the liver, spleen, pancreas and suprarenals are engorged with blood, but the kidneys appear normal. The changes in urinary constituents are very important, but for these the reader is referred to the original paper.

If the animal remains alive after the fourth or fifth day it passes into the sub-acute stage; there is foetid and hypocholic diarrhoea more or less remittent, pulse soft and slightly rapid, dysuria, oliguria choluria, urobilinuria and indicanuria, and there is frequently leucine and tyrosine in the urine also. The sub-acute symptoms may last for several months, and the animal may ultimately pass into the chronic stage which is compatible with perfect health. Animals deprived of the solar plexus have been kept under observation for six or eight months, during which time they have shown no morbid symptoms. As a rule, recovery seems to be complete from six weeks to two months after the operation, and the function of the solar plexus is compensated by some other mechanism.

The study of the solar syndromes of paralysis leads to the conclusion—(1) that the splanchnics play no part in the phenomena

observed, for simple section of these nerves produces nothing similar; and (2) that the solar plexus commands the vaso-motor nerves of the gastro-intestinal tract and its annexed glands.

*The solar syndrome of excitation* in its acute form consists in pain in the epigastrium, constipation and increased arterial tension due to abdominal vaso-constriction. The constipation is due to the inhibition of intestinal peristalsis, and at the same time diminution of intestinal secretion.

The best example of a *solar symptom of irradiation* is seen in the fact first observed by Brown-Séquard, that crushing the solar plexus between the limbs of a pair of forceps produces, almost at once, stoppage of the heart in diastole. This does not take place, however, if the splanchnic and pneumogastric nerves have been divided previously. A violent blow on the epigastrium will often cause syncope or death in sound dogs, but not in animals from which the solar plexus has been removed.

The author endeavours to show that the symptoms found in many abdominal conditions such as peritonitis, lead poisoning, etc., are probably due to an involvement of the solar plexus; e.g. in acute peritonitis the symptoms are not due to the inflammation of the peritoneum, but rather to this acting as an excitant of the solar plexus. Active peritonitis and traumatism of the plexus produce the same solar syndromes.

SUTHERLAND SIMPSON.

#### **THE BLOOD IN EPILEPSY: EXPERIMENTS ON ANIMALS. F. S.**

(220) PEARCE and LE N. BOSTON, *Am. Journ. of Insan.*, April 1904, p. 597.

THE authors consider that a toxin must at times be resident in the blood cells or serum.

In seven cases of epilepsy they found some anæmia of chlorotic type, slight poikilocytosis and some granular basophilia with leucocytosis.

They mention two patients with pernicious anæmia who suffered from fits after being injected with blood taken from the subjects of epilepsy. Rabbits were injected with the blood of epileptics and the authors consider that their results point to epilepsy being a toxæmic process. None of the animals suffered from convulsions, though they showed tremors.

The blood changes included a rise in the percentage of hæmoglobin, leucocytosis and eosinophilia—in one case to the extent of 56 per cent.!

There is no mention of basophils in the records of differential counts, although, in the rabbit, these cells are fairly numerous.

ALEXANDER GOODALL.

**A CASE OF ERYTHROMELALGIA WITH THE POST-MORTEM  
(221) FINDINGS.** ARTHUR S. HAMILTON, *Journ. Nerv. and Ment. Dis.*,  
Vol. xxxi. No. 4, April 1904.

THE case reported is that of a man aged thirty-nine years, of good antecedents and of good general health, who in 1899, after a short illness of uncertain character associated with pain and tenderness over the liver and slight jaundice, commenced to suffer with dull pain and tingling in the left index finger. This disappeared four months later when similar trouble began in the right hallux, which developed into typical erythromelalgia. The whole of the right foot became slowly involved. Subsequently the left foot became similarly affected. The middle toe of the right foot became gangrenous and was amputated.

In 1901 he suffered with general cedema of the trunk and legs and left hand, and this was constantly more marked upon the left side of the body and on the left limbs. He died in an attack of convulsions in December 1901. At the autopsy the following conditions were found. Hypertrophy and dilatation of the heart, slight atheroma of the aorta and chronic valvular endocarditis, old double pleuritis, broncho-pneumonia, enlarged spleen, nephritis, congestion of the liver, adherent dura and cedema of the pia arachnoid.

The muscles of the erythromelalgic regions showed no peculiarity.

In the affected regions there was marked round-celled infiltrations of the derma and subcutaneous areolar tissue. Some of the vessels of the skin showed suppurative arteritis and many were plugged with thrombi.

The medium-sized and small blood-vessels of the affected regions were considerably thickened, the change being chiefly in the tunica media, the interna being but little affected. The lumen was only slightly decreased. In the large arteries the interior showed much obliterative affection, and many of them were thrombosed. The most striking changes were found in the dorsalis hallucis artery of the left foot, of which the muscular coat was greatly and unequally thickened. Inside the elastic coat, which was hypertrophied, was a layer of homogeneous material with a few nuclei scattered through it. Many of the vessels were filled up.

Endarteritis and mesarteritis were general throughout the body.

Obvious degeneration was found in the peripheral nerve of the affected regions.

No changes were found in the spinal cord nor in the posterior root ganglia.

The writer points out that as the records of erythromelalgia

increase, the occurrence of gangrene in association with this disease has been shown to be frequent.

A *résumé* of published works follows, and attention is drawn to the fact that while disease of the blood-vessels has been constantly found, definite changes either in the central or peripheral nervous system have been very unfrequent.

JAMES COLLIER.

**THE CHEMICAL FINDINGS IN THE CEREBRO-SPINAL FLUID (222) AND CENTRAL NERVOUS SYSTEM IN VARIOUS MENTAL DISEASES.** J. H. CORIAT, *Am. Journ. Insan.*, April 1904, p. 733.

AFTER a short review of the literature, Coriat gives the results of his examination of the cerebro-spinal fluid in twenty-nine cases—the fluid being obtained post-mortem by lumbar puncture. The amount varied from 10 c.c. to 150 c.c., the highest figures being found in general paralysis, and supposed to be due to the pial oedema. In all cases the fluid was clear or slightly yellow; in all save two cases it was acid, due to lactic acid. In nine cases a reducing substance was present; this was found to be dextrose, and not pyrocatechin, or any derivative of the cerebrins. Urea was estimated in seven cases. Contrary to the views of the French authors, Coriat holds it of no pathological significance. The largest amount of proteid was found in paralytics, and in other cases of extensive destruction of brain tissue. The proteid was in the form of nucleo-proteid and serum albumin and globulin. The nucleo-proteids are only found in organic pathological conditions. Cholin was absent in only four out of the twenty-nine cases; the largest amounts of cholin seemed to coincide with the largest amounts of proteid.

The author gives a short bibliography.

C. MACFIE CAMPBELL.

## CLINICAL NEUROLOGY.

**SOME NOTES ON DISPENSARY WORK IN NERVOUS AND (223) MENTAL DISEASES.** S. E. JELLIFFE, *Journ. Nerv. and Ment. Dis.*, May 1904, p. 309.

IN 1903, the number of nervous cases treated at the Vanderbilt Clinic was 1860—840 men and 1020 females; the ratio of nervous cases to all cases seen at the Clinic was about 4·5 per cent.

*Mental Diseases.*—196 cases (150 men, 46 women), i.e. 10·5 per cent. These included 75 idiots or imbeciles, 12 stammerers, 1 case of simple mania, 17 melancholia, 5 hypochondriasis, upwards of 30

cases of minor psychoses (28 connected with the menopause), 9 dementia præcox, 26 cases (all men) in initial stage of general paralysis, 7 paranoia, 3 habit-psychoses (morphia or alcohol), 40 alcoholism (only one woman), etc.

*Nervous Diseases.*—1664 cases, 856 "functional" and 808 organic. The *functional* cases included neurasthenia 364 cases, hysteria 79 (3 men), epilepsy 184, choreas 182, tics 21 (tic convulsif 2), spasmodic torticollis 5, paralysis agitans 21 (ages of onset varying from 34 to 67 years).

The cases of *organic* lesion included: (1) Diseases of peripheral nerves, 333 cases, chiefly neuralgia, neuritis and peripheral palsies mainly of traumatic origin. The greater incidence of facial paralysis in the female sex (21 women, 14 men) is noted. Erb's pressure syndrome was present in 20 cases—a strikingly large number which is attributed to "the active co-operation of the department of children's diseases." (2) Diseases of the spinal cord; acute anterior poliomyelitis 21 cases, progressive muscular atrophy 3, tabes dorsalis 35 (31 men), multiple sclerosis 16 (12 men), lateral sclerosis 6 (men), amyotrophic lateral sclerosis 1, syringomyelia 1, myelitis 7, spinal tumour 4, Little's disease 1. The relative affection of the sexes in the cases of multiple sclerosis may be noted. (3) Diseases of the brain, 133 cases, hemiplegia 70 cases. No points of special interest are brought out by these cases, except perhaps the frequency of the diagnosis of cerebral palsies of children—32 cases.

*Muscular Diseases.*—Pseudo-hypertrophy 2 cases; myasthenia gravis 1.

*Miscellaneous Affections.*—Headache 45 cases, migraine 9, alcoholism 40, acroparæsthesiæ 9, exophthalmic goitre 6 (5 women), tremor 8, arterio-sclerosis 17, Raynaud 1, erythromelalgia 1, angioneurotic oedema 1, dermatographism 1, etc.

A. W. MACKINTOSH.

**THE INFLUENCE OF FEVER ON THE PAINS OF LOCOMOTOR**  
(224) **ATAXIA.** C. W. BURR, *Journ. Nerv. and Ment. Dis.*, May 1904,  
p. 320.

BURR remarks on several cases of locomotor ataxia, in which an intercurrent febrile disease greatly aggravated the pains. Two were subjects of malaria. During the malarial attack, from the beginning of each chill until the temperature began to fall, the patients complained of very severe shooting pains—apart from this, the pains were not severe. In two other cases which showed the same marked increase in the severity of the pains, the fever was septic in origin.

His personal experience is against the idea that fever in general



is provocative of tabetic pains, and he thinks "it would seem as if only in those diseases in which chills are associated with fever is there any increase in the ataxic pain," but he has not sufficient data to permit of any positive conclusions being drawn. He refers to the "febrile crises" which have been described as a symptom of locomotor ataxia, and thinks that on account of their rarity they should be regarded rather as a complication.

The question raised—the effect of fever, general or special, on tabetic pains—is certainly one deserving of consideration.

A. W. MACKINTOSH.

**ON TABES AND TABO-PARALYSIS IN CHILDHOOD AND  
(225) YOUTH. JARL HAGELSTAM, *Deutsche Ztschr. f. Nervenheilk.*,  
Bd. 26, H. 3, 1904.**

DURING the last twenty years a number of cases of tabes and general paralysis of the insane in early life have been recorded.

In a comparatively large percentage of the cases of general paralysis in early life, symptoms of tabes have also been observed (31·8 per cent., Alzheimer). In most cases of juvenile tabes and juvenile general paralysis there is evidence of hereditary syphilis. Whilst in the adult general paralysis is more common in males, in juvenile general paralysis males and females are practically affected with the same frequency.

In 39 cases of juvenile tabes recorded in literature, Hagelstam found that 13 were males and 26 females (whilst in adults males suffer from tabes much more frequently than females).

In some cases, syphilis acquired at a very early period of life has been followed by juvenile tabes or juvenile general paralysis.

In 45 cases of juvenile tabes recorded in literature, Hagelstam found, that in more than 25 per cent. there was a history that the father or mother had suffered from tabes, general paralysis, or brain syphilis.

The author records 3 cases. The first was diagnosed as tabes with general paralysis (tabo-paralysis). The patient was 18 years of age. The chief symptoms were ataxia, Rhomberg's sign, loss of patella and tendo Achilles reflexes, muscular hypotonus, inequality of the pupils, loss of pupillary reflex to light, mental symptoms (exaltation), and speech affection.

The second case was that of a patient aged 16. The chief symptoms were inequality of the pupils, Argyll-Robertson pupil on the left side; loss of both tendo Achilles reflexes.

The third case was that of a patient aged 21. The chief symptoms were double optic atrophy, with choroido-retinitis and sclerosis of the choroidal vessels; loss of both knee-jerks and of the right tendo Achilles reflex; loss of sensation to pain at localised

parts of the legs; loss of the vibrating sensation over the right tibia.

From the records of cases of juvenile tabes in medical literature, it appears that ataxia, sensory disturbances and shooting pains are often absent, whilst optic atrophy and bladder troubles are frequently the symptoms which first attract attention.

The following are the more important of the author's conclusions respecting juvenile tabes and general paralysis:—

Tabes (and general paralysis) in childhood and youth develop on a basis of hereditary or early acquired syphilis.

Evidence of hereditary taint is obtained much more frequently than in tabes of the adult; and in a large number of cases the parents have suffered from tabes, general paralysis of the insane, or cerebral syphilis.

In juvenile tabes, females are more liable to be affected than males.

The first symptoms of juvenile tabes are usually noted at the time of puberty, in some cases earlier, in others between 20 and 30 years of age.

A very complete bibliography is added to this article.

R. T. WILLIAMSON.

#### **ON CYTODIAGNOSIS IN TABES AND GENERAL PARALYSIS-**

(226) H. S. FRENKEL, *Monatsschr. f. Psych. u. Neur.*, May 1904 p. 398.

TWENTY-THREE tabetics were punctured; positive cytological result in all, the number of cells being greater the earlier the stage of the disease.

C. MACFIE CAMPBELL.

#### **SOME RESULTS OF THE EXAMINATION OF THE CEREBRO-**

(227) **SPINAL FLUID OF SYPHILITICS.** W. FUNKE, *Arch. f. Derm. u. Syph.*, H. 3, 1904, p. 341.

FUNKE in 40 cases of syphilis—condylomatous, gummatous, hereditary—found only once lymphocytes in the cerebro-spinal fluid. In this case there was atrophy of the optic nerve, with Argyll-Robertson pupil. He agrees with Ravaut that while in organic nervous affections due to syphilis the presence of lymphocytes is fairly constant and of diagnostic importance, lymphocytes are only exceptionally present in the headaches of syphilitics of whatever stage, and are not constantly present in the nervous symptoms of the early stages of syphilis.

C. MACFIE CAMPBELL.

**A CASE OF FRIEDREICH'S DISEASE, COMPLICATED WITH (228) CEREBRAL SOFTENING ; AUTOPSY.** A. PIC and S. BONNAMOUR, *Nouv. Icon. de la Salpêtrière*, mars-avril 1904, p. 126.

J. C. TARDY, age 34, admitted to Perron Hospital, Lyons, in 1897. No hereditary or family tendency. Scarlatina at 4, no bad results. At 9, an undiagnosed illness, perhaps typhoid, leaving him thin, weak and sickly. At 14, attacks of vertigo, no loss of consciousness; cerebellar ataxia. Severe headaches, chiefly frontal, from age of 14 to 20. Troubles of gait, at first in the dark, at 15 by day also; crutches at 20; bed-ridden at 25. Speech first affected at 20. On admission, intelligence good, club-foot, knee-jerk gone on right, ataxia of upper limbs, movement gone in lower, speech affection, cardiac arrhythmia, acuteness of hearing lowered. Least effort caused facial congestion. Both knee-jerks gone in 1900. In March 1902, left hemianæsthesia; probably left lateral hemianopsia. Death in June.

*Autopsy.*—Softening of left temporo-occipital convolutions extending back to cuneus. Softening in optic thalamus, barely involving internal capsule. Sclerosis of pyramidal tracts in bulb down to crossing. Marked degeneration of nuclei of 8th, 10th and 12th pairs. Throughout length of bulb and cord, sclerosis of Goll's columns, best marked in cervical region; sclerosis of Burdach's, best marked in dorsal; sclerosis of crossed pyramidal tract and of lateral cerebellar tract, more marked in cervical and upper dorsal; atrophy of posterior horns, sclerosis of Lissauer's zone and of posterior roots, equally marked all along the cord. Slight sclerosis of Clarke's columns in dorsal cord. Posterior root ganglion showed sclerosis and diminution of fibres. Obliteration of central canal. Meninges thickened. Heart: marked interstitial myocarditis. Liver: marked sclerosis. Kidneys: interstitial nephritis.

J. MACKIE WHYTE.

**ACUTE TRANSVERSE MYELITIS.** DINKLER, *Deutsche Ztschr. f. (229) Nervenheilk.*, Bd. xxvi. H. 3, 1904, p. 248.

THERE are two forms of acute myelitis—the transverse and the disseminated. And of transverse myelitis two types occur, distinct from each other, both anatomically and clinically, one a pure medullary myelitis, the other a meningo-myelitis. The former may, however, develop into the latter.

Cases of acute myelitis, according to Dinkler, may be classified etiologically into two groups—those arising from chill and those due to infective processes. Chill as a cause of disease is but

imperfectly understood, and the possibility is not denied of a superadded infective element in many cases.

He records a case of a man of thirty-six, who had acquired gonorrhœa a year previously, and who for six months had been, in the course of his daily work as a labourer, exposed to wet, standing in water up to the ankles. In him the signs of a myelitis of the lower dorsal and upper lumbar regions developed, and subsequently recovered to a large extent. Gonococci were present in the urethral discharge on admission. Dinkler considers cold to have been the chief exciting cause in this patient, and does not consider the gonorrhœa to have had any necessary connection with the case. As an example of a meningo-myelitis of infective origin, he then relates the somewhat unconvincing history of a case following upon an attack of typhoid fever in a youth of seventeen, and apparently associated with a spondylitis of the lumbar vertebra. This case, though exhibiting a degree of paresis of the limbs and pain in the back and around the trunk, had neither anæsthesia, trophic changes, nor paralysis of sphincters, and the reflexes, superficial and deep, were normal.

The third case is of greater interest. The patient, a healthy man of thirty-four, without syphilitic history, was attacked by a subjective feeling of abdominal pressure, which passed off in a few days. A month later girdle-sensation appeared, lasting twenty-four hours, after which weakness of the lower extremities came on, especially in the right limb, together with anæsthetic incontinence of urine. This in turn passed off, and in a fortnight patient could walk normally, and the bladder had also recovered. A week later, after a long walk, the paresis of the lower limbs reappeared, more marked on the right side, and bladder affection appeared as before. The paralysis was flaccid in type, complete in the right lower limb, partial in the left. There was no loss of sensation to any form of stimulus, though patient complained of a subjective feeling of numbness in both legs, especially the right. The left patellar and Achilles reflex were diminished. The plantar reflex on the right side was extensor in type, on the left flexor; cremasteric reflexes weak, abdominal reflexes absent. There was incontinence of urine, and the bowels were confined. The temperature at first was normal. Soon afterwards right-sided ankle clonus appeared. Some voluntary power reappeared in the right foot and knee. The temperature rose, the right lower limb again became totally paralysed, and involuntary spasms appeared in the limb. Ankle clonus became bilateral, trophic bullæ appeared on the dorsum of the left knee, and priapism developed. Slight diminution of sensibility was observed to all forms of stimuli, from the umbilicus downwards, with a hyperæsthetic zone above. In consideration of repeated rigors and the possibility of a pyogenic cause, an operation was undertaken, and the spinal canal was opened in the lower

dorsal region. No abnormality of the dura or within it was detected, and the wound was closed. Ten days later, bed-sores appeared on both hips, the twitchings of the limbs increased in intensity, and the patient died with a high temperature and coma.

Post-mortem, an appendicitis was found, which had perforated at its tip and ruptured into the large intestine. There was also a myelitis in the dorsal region of the cord, a deep abscess in the left gluteal region, and a right-sided cerebral hæmorrhage which had ruptured into the lateral ventricle.

Microscopically, signs of myelitis were present, most marked in the ninth dorsal segment, but also extending, less intensely, as high as the third dorsal. The vessels were engorged, their walls frequently thickened, sometimes the lumen obliterated. There were numerous capillary hæmorrhages in both white and grey matter. The areas of necrotic softening were irregular in distribution. In each of them, either towards the centre or near the periphery, there was a greatly thickened vessel, usually an artery, whose walls were infiltrated with small round cells. Around this were areas of swollen medullary sheaths, axis-cylinders and glia-fibres, frequently also rows of cells loaded with fat-granules, staining well with Marchi's method. These areas of inflammation were irregularly distributed throughout the affected area of the cord. The pia mater showed slight inflammatory changes.

Dinkler at the time of writing the above (1902) knew of only one similar case recorded in the literature, that of van Engelen. He seems to have overlooked Strumpell's article (*Neurologisches Centralblatt*, 1901, p. 415), and the excellent descriptions in Schmaus and Sacki's text-book. Several similar cases have since been recorded, amongst others one in the *Review of Neurology and Psychiatry* (1903, p. 385), by the writer of this abstract.

PURVES STEWART.

**SYRINGAL HÆMORRHAGE INTO THE SPINAL CORD.** Sir (230) WILLIAM R. GOWERS, *Lancet*, Oct. 10, 1903.

CONGENITAL cavities in the cord, with or without surrounding gliosis, may give rise to no symptoms. Symptoms may be produced in consequence of the distension of the cavities or the growth of the adjacent tissue. The author points out that we can hardly suspect a central gliosis and cavity formation in some cases where the symptoms are slight and not characteristic unless their is a "spina bifida occulta" affording additional evidence of imperfect development. He cites a case with anomalous symptoms in which the scar of a "spina bifida occulta" appeared to afford the key to diagnosis. For a number of years Sir William Gowers has

laid great stress upon the necessity, in cases of acute lesions of the spinal cord, of keeping in view the possibility of hæmorrhage into a pre-existing congenital cavity. In this paper he mentions the symptoms which were present in such a case, and describes the microscopic appearances. Sir William Gowers draws special attention to the very intense pain in the spine which was caused by any movement of the body in this case. He regards severe pain as characteristic of hæmorrhage. Another point which is illustrated by this case is that the onset of the symptoms of syringal hæmorrhage may be less sudden than those of non-syringal hæmorrhage.

Two cases are then described, in both of which the author believes that the symptoms are only to be explained by hæmorrhage into a pre-existing cavity.

The first case was that of a medical man, aged 40, who after experiencing a peculiar feeling of giddiness one night, woke the following morning to find his left arm completely powerless and anæsthetic. The absence of any affection of the face and leg, the complete paralysis of the muscles of the shoulder girdle, including the trapezius, sterno-mastoid and pectoralis major, and the distribution of the sensory loss, made it clear that the paralysis was dependent upon spinal and not upon cerebral disease.

The lesion had evidently been a very severe one, and yet it had evidently damaged the left side of the cord only. The patient had, in addition, a congenital defect of certain movements of the eyes, pointing to a congenital defect of structure in the upper part of the pons on the left side; since such defects in the pons may be associated with similar defects in the spinal cord, it seemed probable that in this case the symptoms were dependent upon a hæmorrhage into a pre-existing cavity. At a later date this patient was operated upon for a condition of the hip-joint closely resembling an arthropathy, an additional corroborative point as to the correctness of the diagnosis.

The second case was that of a man affected with the hæmorrhagic diathesis. When aged 16 years he gave himself a sudden shock by jumping over a box, and the next morning he had lost power and feeling in the legs. When seen by Sir William Gowers twenty years later there was a complete and symmetrical loss of sensation both to touch and pain, corresponding in distribution to the fifth lumbar and all the sacral segments together, with a small area of hyperæsthesia on the outer side of the left foot. The only motor symptom present was some defect of power of flexing the ankles. The motor weakness had, in short, been almost entirely recovered from while the anæsthesia had persisted. There was thickening of the ends of the bones forming the knee-joint quite like that of a tabetic arthropathy, and there were also chronic changes in the hip-

joints. For some years the patient had suffered from pain in the legs, a troublesome ulcer on one toe, and occasional transient swelling of the knee-joints, thought to be due to hæmorrhage.

Sir William Gowers is of opinion that the only condition which will explain this case is a congenital cavity or cavities centrally situated, and extending back in the medial line, or bilateral and symmetrical in the posterior columns.

The third case, which was possibly of the same nature, was that of a girl, 21 years of age, who had suffered from slight weakness of the left leg for eighteen months. A complete paraplegia developed rapidly seven weeks before admission to hospital. Upon the day on which the paraplegia developed there was a great deal of pain in the left leg and spine, also a pain around the abdomen at the level of the umbilicus, which was felt for a few hours from time to time. Upon admission, there was complete paralysis of the lower limbs, with the exception of slight ability to flex the ankles, and fair power in all the intrinsic muscles of the feet. In some muscles of the lower extremities there was an absence of faradic excitability.

There was extensive anæsthesia and analgesia over the lower limbs, with some hyperæsthesia and hyperalgesia in the back above the iliac crest. During the patient's stay in hospital perfect recovery took place in all the affected muscles, a fact which makes it improbable that there had been a primary lesion of the anterior horn. Sensation improved very slowly, much more slowly than motion. "When she left hospital there was only absolute loss of sensation in the hypogastric region and a spot on the outer side of the right foot and inner side of the left leg. The knee-jerks were still absent, the legs could be moved freely and with force in any way as she lay, but it was impossible for her to stand on account of the extreme degree of inco-ordination."

The author's chief object in this paper is to call attention to the class of symptoms which seem to indicate hæmorrhage into cavities.

With regard to treatment, Sir William Gowers insists upon the extreme importance of rest in every case of sudden spinal palsy; if possible the position should be such that the spine is not the lowest part.

EDWIN BRAMWELL.

**A CASE OF PROBABLE CIRROID ANEURISM OF THE CERVICAL (231) CORD.** RAYMOND and R. CESTAN, *Rev. Neurol.*, May 30, 1904, p. 457.

UNDER this title, the authors describe a rare case which recently came under observation at the Charcot Clinic.

Without any antecedent disease, the patient, a man of 17,

began to suffer from pains in the back of the neck and radiating down the right arm. Atrophic paralysis of the right deltoid, biceps, brachialis anticus and supinator longus gradually followed, accompanied by spastic weakness of the rest of the right arm and of the right leg. When examined at the age of 20, there was also found anæsthesia to all forms of sensation corresponding to the lower part of the cervical and upper part of the brachial plexuses. The symptoms gradually spread to the opposite side, but at no time did a Brown-Séquard phenomenon develop. There were no signs of vertebral disease.

An attack of influenza carried off the patient at a time when operation was being considered. At the autopsy it was found that the vertebræ and dura mater were everywhere normal, the latter not being adherent to the subjacent structures. With the theca opened, the cervical region of the cord was found to be surrounded and infiltrated by a veritable network of vessels—arteries, veins and capillaries being hugely dilated. Cross-sections showed that these vessels invaded chiefly the right half of the cord from the level of the pyramidal decussation down to the first dorsal segment. Much destruction of the nervous tissues with gliosis occurred around the dilated vessels, but no trace of recent or old inflammation could be discovered in the blood-vessels.

After discussing the pathology of the condition, the authors conclude that the vascular enlargement was not secondary to a myelitis, and was probably of the same nature as cirroid aneurism.

STANLEY BARNES.

#### ON MULTIPLE SCLEROSIS OF THE BRAIN AND SPINAL CORD.

(232) DINKLER, *Deutsche Ztschr. f. Nervenheilk.*, May 1904, p. 233.

IN this paper the author gives a description of the clinical and microscopical appearances in a case of multiple sclerosis of the spastic paraplegic type. The illness began at the age of twenty-five years, the patient dying eighteen years afterwards of empyœma and sepsis. When she came under observation seven days before death there was complete paralysis of the legs, with increased reflexes and Babinski's sign, retention of urine and fœces, strabismus and diplopia; but there was neither nystagmus, intention tremor, nor alteration of speech, and no sensory disturbances. Although multiple sclerosis was considered, it was impossible to make a certain diagnosis during the lifetime of the patient.

The microscopical examination showed numerous small areas of disease in the brain cortex, as well as larger ones through the greater part of the spinal cord. Within these areas the myelin sheath of the nerve fibrils had undergone fatty degeneration, and in many cases absorption; the axis-cylinders remained for some time unaffected, but later they also underwent degeneration.



These changes in the myelin substance sometimes occurred apart from any area of sclerosis. Many of the ganglion cells of the cord were in a condition of chronic atrophy, whilst those of the cerebral cortex were often encircled by glia cells in a very curious manner. The blood-vessels and membranes showed no characteristic changes. Similar degenerative changes also occurred in the anterior and posterior spinal roots, leading to the formation of numerous little tumour-like swellings in these situations. The peripheral nerves and posterior spinal ganglia were not examined.

A. F. TREDGOLD.

**UNILATERAL LESION OF THE SPINAL CORD, THE RESULT  
(233) OF A STAB WOUND: BROWN-SÉQUARD'S PARALYSIS.**

BYROM BRAMWELL, *Clinical Studies*, Vol. ii. 1904, Part iv., p. 360.

THE patient, a Pole, aged 24, was stabbed in the neck with a small, thin-bladed knife on October 16, 1903. On his admission to the Infirmary on March 22, 1904, a cicatrix measuring  $\frac{1}{2}$  in. by  $\frac{3}{8}$  in. was present half an inch to the left of the middle line just below the apex of the spine of the first dorsal vertebra. The injury was followed by loss of power in the left leg and loss of sensation on the right side of the body (the exact height at the commencement not ascertained); the arm was unaffected. On admission to the Infirmary there was:—

(1) Impairment of motor power in the left leg, the movements of toes, ankle, knee and hip being all affected; exaggeration of the knee-jerks on both sides; knee-clonus and ankle-clonus on the left side; the Babinski's sign on the left side, no toe movement on the right side. The cremasteric reflex was present on the right side, absent on the left; the abdominal reflex was absent on both sides.

(2) No affection of bladder or rectum at any stage of the case.

(3) Loss of all forms of skin sensation (a light touch, pain, heat and cold) on the opposite (right) side of the body below the level of the lesion. This sensory loss was absolute below the level of the third rib, in front, and the apex of the fifth cervical spine, behind; impaired above these levels for about an inch and a half. The upper limit of impaired sensation, behind, corresponded to a point between the apices of the third and fourth dorsal vertebræ, and in front at the level of the second rib. The position of the cicatrix and the level of the anaesthesia on the patient's admission to hospital (March 22, 1904) and on May 5, 1904, are shown in Figs. 1 and 2.

(4) Loss of the sense of pain in the muscles (pinch, powerful faradic currents), and of the sense of pressure on the opposite (right) side below the level of the lesion.

(5) No alteration of skin sensation, either negative or positive (analgesia, hyperalgesia, etc.), on the left side (same side as the lesion).

(6) No impairment or derangement of kinæsthetic impressions and of the sense of movement (the power of appreciating differences in weights, the power of appreciating the direction of passive movements with eyes closed, or of the position of the limbs after passive movements with the eyes closed) on either side of the body. There was no ataxia.

(7) No difference in the size of the palpebral apertures; the right pupil was a shade larger than left.

(8) No difference in vascularity or sweat secretion on the two sides of the face and neck.

*Position of the Lesion.*—There seems to have been a localised lesion of the left half of the cord at the level of the fourth dorsal segment, or perhaps between the point of origin of the third and fourth dorsal roots from the cord. The crossed pyramidal tract on the left side was injured. The fibres carrying skin sensations of all forms and painful sensations from the muscles, from the opposite side of the body, below the level of the lesion, appear to have been completely interrupted. The posterior columns and entering posterior roots were apparently unaffected.

*Conclusions.*—The case seems to show :—(1) That, in this man at all events, all forms of sensory impressions from the skin (touch, pain, heat and cold), and painful sensations from the muscles, completely decussate immediately after their entrance into the cord, and presumably (since there was a lesion of the lateral column and no evidence of a lesion of the posterior column) pass up to the brain through the lateral column. (2) That the upward path through the cord for kinæsthetic impressions is different from that for skin sensations (touch pain, heat and cold) and for painful sensations from the muscles, and is presumably through the postero-internal column (column of Goll). (3) That skin sensations play no part, or no necessary part, in the "sense of movement."

*Explanation of Plate.*—In figure 1 the numbers 1, 2, 3, 4 and 5 point to the apices of the spinous processes of the first five dorsal vertebrae. The letter A points to the upper limit of the absolute, and the letter R to the upper limit of the relative anæsthesia.

Figure 2 represents the results of an examination made by Dr Edwin Bramwell on May 5, 1904, to ascertain the upper limit of the anæsthesia and (cutaneous) analgesia in the case of P. L.

*Tactile Sensibility.*—This was tested with a small camel's-hair paint-brush. The patient's eyes were closed, and each time he felt a touch he was told to answer "Yes." Every application of the brush is recorded in the figure, in which a dot represents a spot at which a touch was felt, while a cross represents a spot at which a touch was not recognised. It will be seen :—

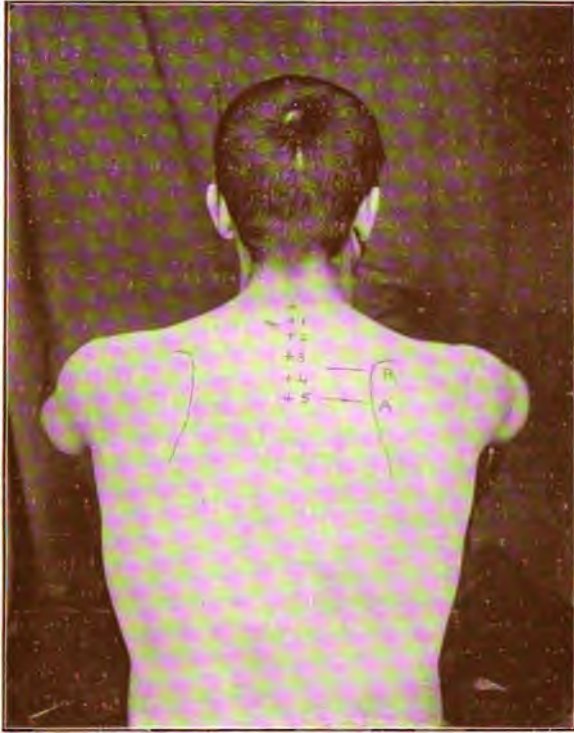


FIG. 1.—Photograph showing the exact position of the cicatrix and the level of the absolute and relative anæsthesia in the case of P. L. described in the text, on March 22, 1904.

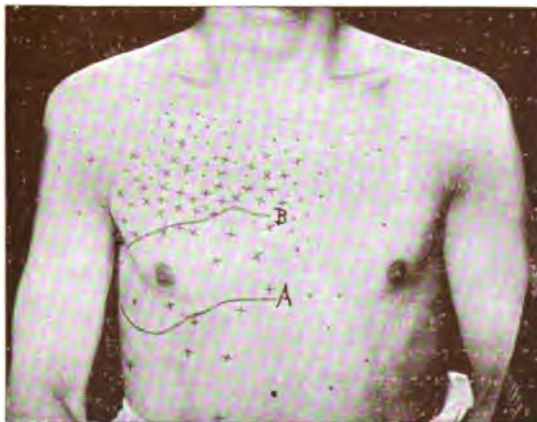


FIG. 2.—Showing the condition of skin sensations, to touch and pain, on the front of the body, on May 5, 1904.



(1) That there are no crosses (no spots at which a light touch was not felt) on the left side of the body.

(2) That below the line B, on the right side of the body, there are no dots (no spots at which a light touch was felt).

(3) That above the line B, on the right side of the body, there are many spots at which a touch was not felt (many crosses): in other words, above the line B, up to the level of the second rib, there is a condition of relative anaesthesia—many crosses and some dots, the crosses decreasing in number and the dots increasing in number from below upwards.

*Cutaneous Analgesia.*—This was tested by drawing the point of a pin in parallel lines up and down the chart. The lower line (A) in the figure represents the level at which the scratch of a pin drawn from below upwards was first recognised as painful. The upper line (B) marks the level at which the scratch of a pin drawn from above downward began to become less painful. In other words, the lower line (A) marks the upper limit of the absolute analgesia; the upper line (B) marks the upper limit of the relative analgesia; the space between the lines A and B represents the area of relative analgesia. Above the line B, painful impressions were normally and vividly felt.

AUTHOR'S ABSTRACT.

#### SPINAL DEFORMITIES—SCIATICA WITH KYPHO-SCOLIOSIS.

(234) H. FORESTIER, *Nouv. Icon. de la Salpêtrière*, mars-avril 1904, p. 88.

M., æt. 40. Previous history unimportant. This man was suddenly seized with tingling in the left buttock followed by pain down the left leg. For seven months he suffered from a moderate degree of pain down the left sciatic nerve, and then the pain became much more intense, the left buttock and thigh began to waste, and he noticed that he stooped.

About a month later he was taken into hospital. He was suffering from constant pain in the left lower limb and tenderness on pressure over the sciatic nerve of that side. The left thigh was somewhat wasted.

The tendon reflexes on the left side were increased, while on the right side the ankle-jerk was absent and the knee-jerk diminished. The plantar reflexes were flexor on both sides, and the abdominal and cremasteric reflexes were absent. Sensation was normal on the left limb but tactile sensation was impaired on the right. The spine showed a marked degree of kyphosis and some scoliosis. Under treatment with the douche and massage he made a rather unexpected recovery in about seven months. This case the author considered to have been one of spasmodic sciatica due to a hæmorrhage in the root of the sciatic nerve, and that a neuro-pathic element superadded gave rise to the deviation of the

vertebral column. He points out the rarity of the occurrence of kyphosis as compared to scoliosis in cases of sciatica.

T. GRAINGER STEWART.

**SPINAL DEFORMITIES—THREE CASES OF RHEUMATIC SPONDYLOSIS AND ANKYLOSIS.** H. FORESTIER, *Nouv. Icon. de la Salpêtrière*, mars-avril 1904.

THIS paper like the previous one is illustrated by a series of excellent photographs.

The cases described are of interest, as they clearly show the rheumatic origin of the condition. The patients were quite free from any trace of venereal or tubercular disease, but each of them had suffered previously from attacks of acute or subacute articular rheumatism which had developed in the course of their daily work while exposed to cold, damp or draughts.

In all the cases the rheumatic change had after a longer or shorter period affected the spine, and it is of interest to note that their occupations were such as to place a certain strain on the back, as two were cobblers and the other engaged in plucking fowls. The period which elapsed between the primary affections of the various joints, and the later involvement of the spine was characterised in all the cases by general pain, backache and girdle pains, and constituted the "période pseudo-névralgique" which has been observed by so many writers. The author points out that unless the rheumatic process is arrested or modified by treatment in this stage, the further stage—that of ankylosis of the spine—is sure to supervene.

Clinically the cases displayed the peculiar attitude so typical of the disease. The head bent forward, the upper spine arched, the lower spine straight, while the lower limbs are always slightly flexed at the knees. Viewed from in front the appearance is not unlike that seen in Parkinson's disease, save that the facial expression is not altered. Their gait is characterised by slight lateral oscillations of the trunk.

All the cases were treated with thermal massage douche. Considerable amelioration of the pains and stiffness resulted from this treatment.

T. GRAINGER STEWART.

**A CASE OF KYPHOSIS OF ARTICULAR OR MUSCULAR (236) ORIGIN.** E. BRISSAUD and H. GRENET, *Nouv. Icon. de la Salpêtrière*, mars-avril 1904, p. 85.

THIS condition developed in a man æt. 37 who had previously suffered from rheumatism and was the suspected subject of pulmonary tuberculosis.

Seven years previous to his admission into hospital he had

suffered from severe pains in the lumbar region. These pains were aggravated on movement and obliged him to remain in bed. For three years he remained in this condition, at the end of which time the pains ceased altogether. Some time later the kyphosis began to develop and gradually became more marked.

On admission to the hospital it was found that the spinal column alone was affected, all the other joints being healthy, and further, that the ankylosis present in the spine was not complete as the curvature diminished both when the patient was lying down and when he was suspended. Another peculiarity of the case was that the thorax was immobile and that the abdominal muscles were firmly contracted, while the spinal muscles were more or less flaccid. The faradic reaction of the abdominal muscles was brisk, while that of the spinal muscles was diminished.

In discussing the case, the authors point out the resemblance of their case to those of rheumatic spondylosis with ankylosis described by Forestier, and also to those cases of kyphosis seen in vine-dressers, differing, however, from the former class in the absence of complete ankylosis, and from the latter in the inability of their patient to straighten his back.

They consider the kyphosis to have been due either primarily to an alteration in the relative tone of the abdominal and spinal muscles, or to articular changes in the vertebral column itself.

T. GRAINGER STEWART.

**A CASE OF CEREBRAL TUMOUR WITH THE CLINICAL  
(237) APPEARANCE OF GENERAL PARALYSIS. E. CORNU,  
*Nouv. Icon. de la Salpêtrière*, March, April, 1904, p. 107.**

CORNU gives the case of a woman of 36 years of age, with no history of syphilis, presenting various symptoms leading to the diagnosis of general paralysis. The evolution of the disease lasted 15 months, beginning with occipital headache, paresis and awkwardness of the limbs, progressive mental enfeeblement. In the course of the illness one noted trembling of the lips and tongue, pupillar inequality, exaggeration of the reflexes, contracture affecting the distribution of the superior and inferior facial: incontinence of urine and faeces. Patient's mental condition was one of pleased childishness, the *Moria* or *Witzelsucht* of German authors. In the terminal stages she had little febrile attacks.

At the autopsy one found a tumour about the size of a walnut of bony nature occupying the point of the right caudate nucleus. The cerebellum was small and especially atrophied on the left side; the right side of the medulla and the right olive were atrophied; right side of the cord was of less volume than the left. Cornu seeks to correlate the clinical facts and pathological data. He does not allow the frontal region to be the centre for the

highest psychical activity, but quotes Gianelli's opinion that when a cerebral tumour presents the syndrome of general paralysis it is probably situated in the frontal lobe. The difficulty in walking and exaggeration of reflexes were probably due rather to the cerebellar lesions than to compression of the internal capsule, and these same lesions explain the muscular paresis, deviation of body and head to right, disturbance of speech and tremor of arm. Compression or irritation of the internal capsule would explain the left facial spasm. The intellectual degeneration, loss of memory and spontaneous activity, are probably related to the chronic meningo-encephalitis so often associated with cerebral tumour; these diffuse lesions and the febrile attacks may be due to toxic products of the neoplasin.

Summing up, Cornu considers that the tumour had existed for a considerable time, with only the localising symptoms, until the time when its toxic or otherwise irritant nature caused the meningo-encephalic lesions giving rise to the more acute symptoms.

C. MACFIE CAMPBELL.

**REMARKS ON THE UNCINATE GROUP OF FITS AND ON**  
(238) **SEVERE SUBCUTANEOUS HÆMORRHAGE OCCURRING**  
**IN EPILEPSY.** WILLIAM G. SPILLER, *American Medicine*,  
Vol. vii., No. 12, March 19, 1904, p. 474.

DR SPILLER describes in this paper a few clinical cases which he regards as instances of the uncinat type of fit, first described by Hughlings Jackson.

In one case, when the attack is coming on, the patient sees a bright light in front of her, objects appear strange and out of place; sometimes they are large and sometimes small, and she has the taste of raw, unsalted beef in her mouth.

Another patient had attacks which began with a creeping sensation in her left upper limb, which seemed to ascend the limb gradually, and to involve the left side of the tongue, so that the tongue felt thick. She was then obliged to sit down, and seemed to be unconscious, although she heard all that was said to her, but she could not speak. She saw everything about her during the attack, but vision was blurred. She had a "grassy taste" at the time of the attack.

A third case was that of a man who had taken three convulsive attacks during the five months before he was first seen by Dr Spiller. His wife stated that for a year and a half before the first fit he had had what she termed "swallowing spells," in which he made the noise and movement of swallowing and rubbed his fingers together. The attacks lasted a minute or two. His wife stated that during these turns he was unconscious, that his face did not turn blue, that he had no peculiar taste in his mouth and no



chewing or spitting movements, and that he did not smack his lips. This patient had severe sub-cutaneous hæmorrhages of the face and right upper limb after a convulsive attack. A neuritis of the right arm followed the attack, and appeared to be due to hæmorrhage in the region of the brachial plexus.

Another case is described in which a right hemiplegia and disturbance of speech developed during the status epilepticus, in consequence of a cerebral lesion, possibly hæmorrhage or thrombosis.

EDWIN BRAMWELL.

**APHASIA WITH LEFT-SIDED HEMIPLEGIA IN RIGHT-  
(239) HANDED INDIVIDUALS.** H. SENATOR, *Charité-Annalen*,  
xxviii., Jahrg. 1904.

THE author describes the case of a right-handed woman, aged 38, who was admitted to the Charité suffering from aortic incompetence with associated failure of compensation. While in hospital she suddenly developed, on July 13th, a left hemiplegia involving the face, arm and leg, with anæsthesia of the same side of the body. In addition, the patient was aphasic. She made no attempt to shake hands when told to do so, and the only words she made use of were, "Ach Gott! Ach Gott!"

On July 16th she showed her tongue upon command, and used some unintelligible words. She could not be persuaded to attempt to write anything, and she appeared to be unable to read. During the next two or three weeks at times she voluntarily made use of a few words; the aphasic condition showed little improvement, however, and the hemiplegia remained much *in statu quo*.

On August 13th the patient died, and at the post-mortem a clot was found in the right middle cerebral artery, and there was an extensive softening of the right temporo-sphenoidal lobe and neighbourhood of the Sylvian fissure on the same side.

The case is an instance of aphasia resulting from a lesion of the right hemisphere in a person who was right-handed. It was not ascertained whether any of the patient's ancestors were left-handed.

Senator discusses "crossed aphasia" and aphasia associated with left-handedness, and suggests the following classification for speech defects of this nature:—

1. Aphasia in complete left-handedness (lesion in the right hemisphere).

2. "Crossed aphasia" of Byrom Bramwell (lesion in the right hemisphere).

(a) In left-handed people in whom the right half of the brain has been trained for writing and speech.

(b) In right-handed people in whom, in consequence of an hereditary tendency to left-handedness, the right half of the brain is used for writing and speech.

3. Aphasia with left-sided hemiplegia, in cases of defective pyramidal decussation (lesion in the left half of the brain).

EDWIN BRAMWELL.

**SENSORY DISTURBANCES IN NEURASTHENIC AND MELANCHOLIC STATES.** M. DUBOIS, *Journ. de Neurol.*, May 5, 1904.

DUBOIS considers that undue importance is laid upon the existence of sensory troubles as helping to diagnose certain incipient cases of general paralysis from simple neurasthenia or melancholia. He gives shortly three cases of doubtful diagnosis where such sensory troubles were present, but in which the progress showed that general paralysis was not in question, but a simple neurasthenia or melancholia.

C. MACFIE CAMPBELL.

**THE FORCE OF THE TENDON-REFLEXES AND THEIR CHANGE (241) IN HEMIPLEGIA.** K. PÁNDY, *Neurolog. Centralbl.*, May 16, 1904.

IN a case of epileptic dementia which developed complete hemiplegia after a series of fits, Pándy found reduction of the knee-jerk on the paralysed side, so that it was only able to raise a weight of 3 k.g. in contrast to 10 k.g. which the knee-jerk of the opposite limb could raise, while the reflex was also slower and the secondary contraction of the flexors, which under normal conditions fixes the limb, did not take place. In this observation the author finds support to the theory formerly enunciated by him, that the apparently brisk and exaggerated reflex movements of the paretic side in hemiplegia are, when more closely examined, found to have undergone a change analogous to the affection of the voluntary movements, and further that the paths of voluntary innervation and the reflexarcs are identical. The increase of the reflexes in the older cases of hemiplegia are explained as due to the persistent stimulation of the damaged cortico-spinal fibres, and secondly to the restriction of distribution of the reflex-processes. The author recommends that in the study of the tendon reflexes attention should be given to (1) the relation of the force of the reflex movement to the strength of the quadriceps; (2) the rapidity of the movement; (3) the range of movement; (4) the force of the movement.

He mentions another form of knee-jerk which is present in decerebrate dogs and in recent cases of hemiplegia: the reaction to the tap is brisk, but the leg suddenly falls again quite flaccid and begins to swing about as an inert and powerless object.

Bastian's theory of the absence of the knee-jerks in total transverse lesions of the cord is accepted as universal, provided that the lesion is not of an initiative nature.

Some points in the paper are suggestive, but much is open to

criticism, especially the assumption of persistent stimulation, a persistent positive symptom from a primarily negative lesion.

GORDON HOLMES.

**ON THE CREMASTERIC REFLEX AND THE SUPERPOSITION (242) OF REFLEXES.** STEINER, *Deutsche Ztschr. f. Nervenheilk.*, Bd. xxvi. H. 3, 1904, p. 285.

THIS writer examined a series of 100 males, mostly soldiers, all of them free from nervous disease, by pricking with a needle the skin of the thigh. In 92 per cent. the cremasteric reflex was brisk, in 3 per cent. feeble, and only absent in 5 per cent. Of the 5 cases in which it was absent, 2 exhibited a distinct reflex contraction in the inguinal region (Geigel's inguinal reflex). This inguinal response is to be considered a sub-variety of the cremasteric reflex. The resulting proportion, 97 per cent., at which he thus arrives, is much in excess of the figures given by various other observers, notably by Schönborn and by Geigel, both of whom estimate its frequency at about 30 per cent.

The reflex was sometimes unequal on the two sides. Sometimes a unilateral stimulus produced a bilateral response. In one case the reflex was also elicited by psychical stimulus. The patient was watching the physician when preparing to prick the skin of the thigh, and before the pin reached the skin the testicle became drawn up. The phenomenon no longer occurred when the subject's eyes were bandaged. In no case was an exhaustion of the cremasteric reflex observed after repeated stimulation, contrary to the experience of Geigel. Steiner, however, does not deny the possibility of such an occurrence. The area of skin from which the reflex may be elicited was generally the whole inner and anterior surface of the thigh. It rarely reached down to the knee, but usually stopped about a hand's breadth above it. In a few cases the area extended below the knee to the upper part of the leg. Of the thigh area, the most sensitive part was the middle and inner portion. The nerve areas implicated are stated to be those of the external, middle and internal cutaneous, the ileo-inguinal, and the iliac branch of the ileo-hypogastric. (No reference is made to the genito-crural nerve!)

Steiner further remarks that the range of movement in the testicle varies according to the length of cord swinging free. Individuals in whom the testicles hang low tend to have a more marked reflex than those in whom the gland is habitually closely apposed to the external abdominal ring. (This is not in harmony with the well-known briskness of the reflex in little children, compared with its sluggishness in old men.)

No constant connection was observed between the intensity of the cremasteric and that of the patellar reflex.

The inguinal reflex and its connection with the cremasteric are

then discussed. Inasmuch as the cremaster muscle is but a specialised part of the internal oblique, the anatomical unity of the two muscles is evident. Many cases show both reflexes simultaneously when the skin of the thigh is stimulated. Steiner states that both reflexes are but a portion of the general abdominal reflex. In some cases, on starting the stimulus near the knee and proceeding gradually upwards, cremasteric and inguinal reflex appear together. Mounting up the inner side of the thigh, the meso-gastric reflex, and afterwards the epigastric, are superadded, the latter on reaching a level slightly higher than that of the lower edge of the testicle. This phenomenon he desires to term the "superposition of reflexes," and explains it on the hypothesis of sensory paths reaching two or more different levels in the cord. In one case of injury to the lumbar region of six years' standing, in which some weakness of the lower limbs remained, without bladder affection or sensory changes, the left knee-jerk was brisker than the right and the left calf slightly wasted. In this patient the "superposition of reflexes" was absent on the left side and present on the right, though the abdominal and cremasteric reflexes could both be elicited separately on the left side. Steiner therefore concludes that the connection between these two reflexes was interrupted, and that the interruption was within the cord.

In conclusion, the writer states that since his paper was written (in 1902) he has directed more attention to the above points, and promises "several new things," to be published later. A corollary is to be noted, in which he states that in fat and flaccid abdominal walls the phenomenon of "superposition" is frequently indistinct or absent.

PURVES STEWART.

**SOME GRAPHIC OBSERVATIONS OF ANKLE CLONUS.** AUGUSTUS (243) ESHNER, *Journ. Am. Med. Ass.*, May 7, 1904, p. 1202.

THE question as to whether ankle clonus can be reinforced, in a similar manner to the knee-jerk, by motor influences, was studied by kymographic tracings, with the result, that increased frequency of movement during the reinforcement was shown in two out of six cases examined.

The variations in the frequency of the clonus for all investigated cases was between 5.8 to 8 per second.

G. W. HOWLAND.

**ON THE RELATION BETWEEN TRIGEMINUS NEURALGIA (244) AND RECURRENT CORNEAL EROSION.** M. BARTELS, *Münch. med. Wchnschr.*, April, p. 746.

RECURRENT corneal erosion has, on account of its peculiarity, received considerable attention, and several widely diverging

explanations. The writer gives a long account and criticism of the theories of Szili that it is due to a peculiar friability of the epithelium, which allows it to be rubbed off by the lids, and of Peters that the cause is an œdema of the epithelium, brought about through neuritis of its nerve endings.

The writer agrees so far with Peters. He prefers to attribute the friability of the epithelium to an abnormality of epithelial formation, as he has carefully examined a case without detecting any sign of œdema, but he finds more difficulty in accounting for the reason of the attacks than for the manner of loss of surface.

He wishes to draw a sharp distinction between the results on the epithelial surface of neuritis and of the functional condition of neuralgia, in which he states there is not necessarily any organic change. These recurrent corneal erosions he desires to attribute to trigeminal neuralgia. He supports his views by the records of three cases, and suggests that, in all cases of recurrent corneal erosion, if the fifth nerve be carefully examined, other evidences of neuralgia will probably be found.

He includes in the paper suggestions upon the treatment of the recurrent erosion and facial neuralgia. JOHN D. COMRIE.

**ON THE AFFECTION OF THE MUSCULUS ORBICULARIS  
(245) OCULI IN CORTICAL AND SUB-CORTICAL FACIAL  
PARALYSIS.** W. v. BECHTEREW, *Centralbl. f. Nervenheilk. u.  
Psychiat.*, May 15, 1904, p. 305.

THE writer does not consider that the escape of the upper portion of the face in cortical and subcortical lesions has been finally explained, though he considers that the theory of bilateral representation of the upper branch of the facial nerve in the cortex cerebri is best founded.

Experiments on monkeys and dogs convinced him that the centre for the upper part of the facial nerve is situated in the hinder part of the second frontal convolution, separated in some measure from the centre for the lower part of the nerve. Raising and lowering of the eyelids appeared always as a bilateral phenomenon when the cerebral cortex was stimulated. As to shutting of the eyelids tight, in animals this appeared to be due to double innervation, the crossed influence being specially powerful.

In man cortical and subcortical lesions show an involvement of the orbicularis oculi. The person can, as a rule, shut both eyes at once, and he can shut the eye corresponding to the lesion while he holds the other open, but not *vice versa*. The reflex closure of the lids is unaffected.

This sign, which attracted the attention of French observers, the author considers of importance, as indicating, in lesions above the seventh nucleus, involvement of the paths or centre of the upper part of the facial nerve.

He notes that he has observed it in a case of old-standing right-sided hemiplegia, with apparently complete recovery, in which indeed the sign in question was the only symptom left. Also that he has observed the same symptom in peripheral facial paralysis.

JOHN D. COMRIE

**A CASE OF CONGENITAL HIGH POSITION (HOCHSTAND) OF (246) THE SCAPULA, WITH A DIGEST AND CLASSIFICATION OF HITHERTO REPORTED CASES.** RUSSELL A. HIBBS and H. CORRELL-LOEWENSTEIN, *Archiv. f. Orthopädie, etc.*, Bd. 11, H. 1, 1904, p. 40.

SPRENGEL'S shoulder, as it is often called, is a deformity of no little interest to the neurologist.

Drs Hibbs and Correll-Loewenstein have collected from the literature 60 cases; these they classify in four groups:—

1. Cases with a bony connecting bridge between the scapula and vertebral column (11 cases).

2. Cases with complete absence of one or more muscles of the shoulder girdle (5 cases).

3. Cases with a long arched (übergebogenen) supra-spinal part of the scapula (13 cases).

4. Cases without bony outgrowths, with a normal or small scapula and shortened or otherwise defective muscles (31 cases).

The X-rays are of value in recognising those cases which fall into groups 1 and 3, where the obvious treatment is resection of the bridge or process.

The exact muscular defects which may produce this malposition of the scapula in cases where there is no bony process are not considered in this paper, in which the subject is approached rather from the surgical standpoint.

EDWIN BRAMWELL.

## PSYCHIATRY.

**NEURASTHENIC MELANCHOLIA.** M. FRIEDMANN (of Mann-(247) heim), *Monatsschr. f. Psych. u. Neur.*, April-May 1904, p. 301.

FRIEDMANN does not mean a combination of neurasthenia and melancholia, which too is comparatively rare, although in melancholia neurasthenic symptoms are often present; nor is he discussing the question of the relation of the neurasthenic constitution to mental unsoundness. He brings under neurasthenic melancholia or pseudomelancholia a group of cases symptomatically melancholia, but essentially neurasthenia, as seen by its etiology and treatment. It is an exhaustion neurosis. Friedmann mentions another group where neurasthenia comes into contact with the psychoses. These are cases symptomatically neurasthenia, but

running a periodic course. To bring this group under manic-depressive insanity is to overlook the mental soundness of the neurasthenic. Leaving these circular neurasthenias, the author turns to the other group of neurasthenia difficult to distinguish from melancholia. The general problem is, what psychopathic syndromes can develop out of nervous exhaustion? By what criterion can we distinguish them from the psychoses? The question of the fundamental difference between neurasthenia and a psychosis may be split into two. (1) For the majority of neurasthenic conditions is there sharp differentiation from a psychosis? (2) Are the transition forms vague, or can we of such determine which characters are due to neurasthenia, which to the psychoses? The author gives affirmative answers, and gives his criteria to differentiate the two. We have first the etiology and clinical course. In neurasthenia the condition is caused by real worries, the removal of which have marked beneficial value; secondly, the critical faculty of the neurasthenic is not attacked. He recognises his illness, tries to react against it. Nervous exhaustion is a functional disturbance with the physical and psychical symptoms of fatigue. In the latter there are (1) a general feeling of malaise in many degrees; (2) psychic hyperæsthesia; (3) difficulty of attention, judgment and volition.

In the neurosis the depressed feeling is not primary, but is due to objectively based anxieties acting on a hyperæsthetic individual. The delusions of the psychosis are absent—obsessions are present. While the removal of the anxiety improves the neurosis, the psychosis is unaffected. While the neurasthenic remains in his core the same individual, able to judge his abnormal state, the melancholic has become a different personality.

In 98 per cent. of cases of neurasthenia the above criteria are sufficient to establish the diagnosis; in 2 per cent. we have difficult intermediate forms.

We may have: (1) a group clinically like circular insanity—doubts and obsessions, without obvious cause, rising and disappearing; (2) a group symptomatically melancholia, not only running a more violent course than a neurasthenia, but with loss of the faculty of judgment. This latter group may be again subdivided into three groups: (a) strong and partly independent depression calls up typical delusions of exaggerated, not senseless nature; (b) whole mind is dominated by terror; judgment and self-command are lost: here there is danger of suicide from secondary delusions; (c) stuporose conditions. The author gives a general division of neurasthenia, and shows the diagnostic points in the various forms as against melancholia. In the three groups mentioned above he holds that it is not a question of nervous strain but of psychic strain. These patients have a peculiar mental disposition disposed to extremes. A sufficient psychic stimulus may with them develop serious psychopathic traits.

With such dispositions he connects other normal phenomena—outbursts of fanaticism, popular waves of feeling, the *Weltschmerz* of the youth, the enthusiasm of the girl, the depression of the climacterium. In summing up he insists on the recognition of this group, etiology and course being of great help to the diagnosis. Treatment away from the family, but not in an asylum, is recommended. Medico-legally one must note such cases have still present their faculty of judgment. C. MACFIE CAMPBELL.

## TREATMENT.

**ERGOTHERAPY AND PSYCHOTHERAPY: A PSYCHOLOGICAL (248) THEORY OF THE TREATMENT OF THE INSANE BY MEANS OF WORK.** M. I. BIANCHINI, *Nouv. Icon. de la Salpêtrière*, mars-avril 1904, p. 136.

BIANCHINI insists upon the benefits in most types of insanity of suitably chosen forms of manual labour, and gives the statistics of the patients so treated in his own institution. In the insane consciousness is distorted, not abolished: useful work with a definite aim is no longer spontaneously done, but can be attained by means of re-education and mental suggestion, and this activity has, if not a curative, at least a preservative influence on the intelligence.

C. MACFIE CAMPBELL.

**THE TREATMENT OF MORPHINE HABIT BY HYOSCINE.** J. M. (249) BUCHANAN, *Am. Journ. Insan.*, April 1904, p. 635.

IN the treatment of morphine habit, the methods formerly in vogue of sudden withdrawal, or quick reduction, or tapering off, were unsatisfactory. Much more rapid and better results can be obtained by the use of hyoscine hydrobromate, and that too with greatly reduced suffering to the patient. The dose should be small at first,  $\frac{1}{100}$  gr., gradually increased to  $\frac{1}{10}$  gr. At first sleep is induced, but after a few doses the patient becomes restless and then delirious.

If possible—it is not essential—the patient is kept under observation for a few days, and the amount of morphine and cocaine reduced to what will keep him comfortable. Before hyoscine is commenced, 6 to 8 grs. calomel, with a vegetable cathartic, are given at night, and followed by a saline in the morning. The usual morning dose of morphine is given, but in the afternoon, when the patient calls for it again, hyoscine is substituted; and from this time onwards, for from 36 to 40 hours, he is kept under its influence,  $\frac{1}{100}$  to  $\frac{1}{10}$  gr. being given every 2 or 3 hours, so as never to completely stupefy him. He should be kept in bed with constant nursing. If weakness,



general or cardiac, shows itself, strychnine and, if needful, a little morphine and codeine are given. Some patients may require for a time a hypnotic, such as trional or chloral hydrate, with sodium bromide. It is important to see that during the treatment the patient takes a sufficient quantity of milk and water.

If large doses of sodium bromide are given before the treatment is commenced, or if larger doses of hyoscine at shorter intervals are administered, there will be less nervousness, and the time may be passed in sleep. One patient, who had the hyoscine administered while he was under the influence of a large dose of morphine, slept through the treatment.

The after management consists in tonics, nourishing diet, and rest.

It is not claimed that hyoscine is a specific for morphine habit, but a "safe, certain and painless method of treatment." The patient does not miss the morphine when under the hyoscine, nor crave for it at the conclusion of treatment. The writer has found the method most satisfactory in the twelve cases in which he has used it. Dr C. C. Stockard of Atlanta has treated over 300, and Dr G. E. Patty of Memphis about 500 cases, with most gratifying results.

The best antidote for hyoscine poisoning is strychnine and atropine.

A. HILL BUCHAN.

**THE SO-CALLED "GOLD CURE" OF INEBRIETY.** S. BLACKWELL (250) FENN, *Brit. Med. Journ.*, April 30, 1904, p. 1008.

THE method is briefly as follows:—The patient on entering the institution is at once supplied with his favourite beverage if he wants it, and he is subjected to no restraint. A generous diet is given. After thorough medical examination the "cure" begins and consists in:—(1) Hypodermic injections of a solution containing daturine sulphate, gr.  $\frac{1}{160}$ , or atropine, gr.  $\frac{1}{160}$ , and strychnine sulphate, gr.  $\frac{1}{160}$ , the doses being regulated by the patient's condition. (2) Medicine given internally every two hours during the day and evening, generally a mixture containing chloride of gold and sodium, gr.  $\frac{1}{16}$ , ammonium chloride, gr. i, aloin, gr.  $\frac{1}{16}$ , fluid extract of viburnum, ℥x, and tincture of cinchona, ℥xl, the aloin being omitted if diarrhoea occur. Small frequent doses are better than larger ones at longer intervals. If the patient insist on taking alcohol after four or five days, emesis is induced by adding ipecacuanha to his mixture. If the medicines produce gastro-intestinal disturbance, the prescription is changed. Tincture of capsicum, fluid extract of kola, and ammoniated tincture of valerian are drugs frequently used. Insomnia and neurasthenia are carefully attended to. (3) Hydrotherapeutic measures in form of various packs, vapour and other baths. The moral effect of institution life, influence of doctor, etc., are important factors in the cure. The

patient is stimulated to break from his past and look at life in a new way.

In most cases 4, in exceptional cases 5 weeks, are required for cure; very rarely 6 or 8 weeks are needed.

Failures are not numerous. As to percentage of permanent cures, Dr Wolfenden's statistics in Australia would give 80 per cent. as remaining total abstainers after a period of 1 to 8 years; the writer's experience is that 60 per cent. do so.

A. HILL BUCHAN.

**THE TREATMENT OF INEBRIETY BY ATROPINE. C. A. (251) M'BRIDE, *Brit. Med. Journ.*, April 30, 1904, p. 1006.**

THE writer believes that inebriates can generally be treated on a short term system of six weeks. Atropine has a specific action in these cases.

In an adult with no delirium or serious complications the author gives:—(1) Atropine sulphate, gr.  $\frac{1}{16}$ , thrice daily, the dose being gradually increased till physiological effects manifest themselves. Generally  $\frac{1}{8}$  gr. will be the maximum dose. (2) Strychnine nitrate at same time subcutaneously, beginning with  $\frac{1}{16}$  gr. and rising generally to  $\frac{1}{8}$  gr. (3) Fluid extract of red cinchona bark every three hours by the mouth. This routine treatment may require modification in certain instances, and the exhibition of the drugs must be conducted by the medical attendant personally. Warm baths, special dietary, and regulated exercise are valuable auxiliaries. When delirium is threatened, other remedies may be required. It is generally well to give a brisk purge at first. Bovril containing plasmon may be freely used.

Dr M'Bride has used these remedies for over thirteen years with much success in several hundred cases, not merely in the case of alcoholism, but where there was abuse of cocaine and morphine. From the remarkable recoveries he has witnessed in some apparently hopeless cases, he believes that degeneration changes play but a small part in producing the mental deterioration obvious in chronic alcoholics, and would look on such as more largely due to the continued presence of alcohol in the body. Cases illustrative of this line of treatment in different types of alcoholism are given.

A. HILL BUCHAN.

**ON THE METHODS OF APPLICATION OF HIGH FREQUENCY (252) CURRENTS. CLARENCE A. WRIGHT, *Med. Electrol. and Radiol.*, May 1904, p. 191.**

IN continuation of his previous paper the author after discussing, and pointing out various advantages in connection with, certain forms of apparatus, states his conviction that Reus' cones are, *par excellence*, the apparatus for all local applications.

The physiological effects of local applications are to be divided into three classes—those on (a) the contractile tissues, (b) the sensory nerves, (c) the skin.

Under heading (a) it is pointed out that while no idio-muscular contractions occur when the oscillations of high frequency are established, yet momentary local contractions are produced through the passage of the discharge currents when a conductor of small capacity is connected with the free upper terminal of a resonator or cone and approached to the surface of the skin.

These effects are due to the static discharges and not to the dynamic properties of the currents, although it is to be remembered that the dynamical effects of the currents will eventually produce a myasthenia if the session be sufficiently prolonged.

While various explanations are put forward by Cæsare, Hance and Texeira in connection with the effect on the sensory nerves of an impression of local warmth, the true explanation seems to be that given by Reus, that it is due to the heat generated in the skin by reason of its great resistance to the passage of the current. As regards the degree and extent of anæsthesia produced by local applications, many conflicting reports by various authorities are cited.

The author considers that the degree of anæsthesia produced varies with (a) the duration of the session, (b) the character of the discharge, (c) the tension and density of the current, (d) the method of electrification of the patient, (e) the resistance of the skin at the seat of application.

A. DINGWALL FORDYCE.

## Bibliography

### ANATOMY

- BUMKE. Ein Beitrag zur Anatomie der Pyramidenbahn. *Neurol. Centralbl.*, Mai 16, 1904, S. 436.  
 STERZI. Die Blutgefäße des Rückenmarks. J. F. Bergmann, Wiesbaden, 1904, M. 18.60.

### COMPARATIVE ANATOMY—

- KRAUSE und KLEMPNER. Untersuchungen über den Bau des Centralnervensystems der Affen. Das Nachhirn vom Orang Utan. G. Reimer, Berlin, 1904, M. 3.  
 RAMON Y CAJAL. Textura del sistema nervioso del hombre y de los vertebrados. Fasc. 7. Moya, Madrid, 1904.  
 CZARNEICKI. Sur l'aspect extérieur des dendrites des cellules nerveuses des tubercules quadrijumeaux antérieurs et postérieurs chez les vertébrés supérieurs. *Nouv. Icon. de la Salpêtrière*, mars-avril 1904, p. 100.

### METHODS.

- RAMON Y CAJAL. Algunos métodos de coloracion de los cilindros-ejes, neurofibrillas y nidos nerviosos. *Trab. del laborat. de la Univer. de Madrid*, T. iii. 1. 1, 1904.

## PHYSIOLOGY

- HENRI VEDIE. Théorie de la fonction ventriculaire du système cérébro-spinal. Maloine, Paris, 1904, 1 fr.
- ADAMKIEWICZ. Die wahren Centren der Bewegung. *Neurol. Centralbl.*, Juni 16, 1904, S. 546.
- CHARPY. De l'action de la volonté sur les muscles. Marqués et Cie, Toulouse, 1904.
- E. TREVES. Risposta alla memoria del prof. E. Belmondo dal titolo: "Curva micrografica e curva ergografica." E. Belmondo. Replica. *Riv. di Patolog. nerv. e ment.*, Vol. ix., 1. 5, 1904, p. 237.
- WILHELM NEUTRA. Ueber Ermüdungsphänomene auf dem Gebiete der Vibrationsempfindung. *Neurol. Centralbl.*, Juni 1, 1904, S. 510.
- UEKKÜLL. Studien über den Tonus. II. Bewegungen der Schlangensterne. *Ztschr. f. Biol.*, Bd. xlv., H. 1, 1904, S. 1.
- KIESOW. Nochmals zur Frage nach der Fortpflanzungsgeschwindigkeit der Erregung in sensiblen Nerven des Menschen. *Ztschr. f. Psychol. u. Physiol. d. Sinnesorgane*, Bd. 35, H. 2, 1904, S. 132.
- W. STERNBERG. Zur Physiologie des süßen Geschmacks. *Ztschr. f. Psychol. und Physiol. d. Sinnesorgane*, Bd. 35, H. 2, 1904, S. 81.
- SCHOEN. Paradoxes Doppelsehen. *Ztschr. f. Psychol. u. Physiol. d. Sinnesorgane*, Bd. 35, H. 2, 1904, S. 134.
- HILBERT. Versuch eines Systems der physiologischen Farbenempfindungen nebst einem Beitrag zur Kenntnis derselben. *Klin. Monatsbl. f. Augenheilk.* Mai 1904, S. 405.
- JEAN BECQUEREL et ANDRÉ BROCA. Modifications de la radiation des centres nerveux sous l'action des anesthésiques. *Arch. d'Elect. méd.*, juin 10, 1904, p. 414.
- CASTEX. Nouveaux dynamomètres pour le membre supérieur. *Journ. de Physiol. et de Path. gén.*, mai 15, 1904, p. 417.

## PATHOLOGY

- BIKELES. Zur Frage der Regeneration im Rückenmark. *Neurol. Centralbl.*, Juni 16, 1904, S. 559.
- EINAR SJÖVALL. Ueber die Beziehungen zwischen Verbreitungsgebiet des Krampfes und Localisation der anatomischen Veränderungen bei experimentellem Tetanus. *Neurol. Centralbl.*, Juni 1, 1904, S. 498.
- THEODOR KAES. Ueber Markfaserbefunde in der Hirnrinde bei Epileptikern, besonders in der äusseren (zonalen) Associationschicht. *Neurol. Centralbl.*, Juni 1, 1904, S. 504.
- DADDI. Ricerche sulla rabbia. *Riv. crit. di clin. med.*, Anno v., No. 22, 1904, p. 349.

## CLINICAL NEUROLOGY AND PSYCHIATRY

## GENERAL—

- SIR WILLIAM GOWERS. Lectures on Diseases of the Nervous System. Second Series. J. and A. Churchill, London, 1904, 6s.
- M. ALLAN STARR. Organic Nervous Diseases. Baillière, Tindall & Cox, London, 1904, 25s.
- F. X. DERCUM. Mystic Medicine. *Journ. Nerv. and Ment. Dis.*, June 1904, p. 411.
- C. K. MILLS. Neurology in Philadelphia from 1874 to 1904. *Journ. Nerv. and Ment. Dis.*, June 1904, p. 353.
- F. W. LANGDON. Cardio-vascular and Blood-states as Factors in Nervous and Mental Diseases. *Cincinnati Lancet-Clinic*, May 7, 1904.
- INGLERANS. L'hérédosyphilis du système nerveux. *Gaz. des Hôp.*, mai 21 and 28, 1904, p. 573.

## MUSCLES—

- HOLTZAPPLE. Family Periodic Paralysis. *Am. Med.*, April 30, 1904.

## PERIPHERAL NERVES—

- STEINERT. Zur Kenntnis der Polyneuritis der Tuberkulösen. *Beitr. z. Klinik d. Tuberkulose*, Bd. 11, H. 4, 1904, S. 347.
- DEJERINE et EGGER. Un cas de névrite radiculaire sensitivo-motrice généralisée à marche chronique. *Rev. Neurol.*, juin 15, 1904, p. 525.
- BERGER. De la neuro-fibromatose. *Arch. gén. de méd.*, mai 31, 1904, p. 1367.

- Landry's Paralysis.**—L. HARRISON METTLER. Acute Ascending Paralysis and Landry's Paralysis. *Journ. Am. Med. Assoc.*, May 14, 1904, p. 1267.  
**Herpes Zoster.**—PAUL FABRE. Zur la séméiologie et le diagnostic du zona. *Journ. des Prat.*, mai 21, 1904, p. 321.  
 PICK. Statistisches vom Herpes zoster. *Prag. med. Wchnschr.*, Mai 5, 1904, S. 219.

## SPINAL CORD—

- ANDRÉ BROCA et A. ZIMMERN. Étude de la moelle épinière au moyen des rayons N. *Arch. d'Élect. méd.*, juin 10, 1904, p. 411.  
**Meningitis.**—C. L. DANA and RAMSEY HUNT. Tuberculosis of the Spinal Cord, with Reports of Cases of Tuberculous Myelitis and of Pachymeningitis. *Med. News*, April 9, 1904.  
**Tabes.**—MAURICE FAURE. Prognostic actuel et thérapeutique nouvelle du tabes. *Gaz. des Hôp.*, juin 14, 1904, p. 669.  
 BLENCKE. Ein Beitrag zur Arthropathie bei Tabikern. *Ztschr. f. orthopäed. Chir.*, Bd. 12, H. 4, 1904, S. 632.  
 MAURICE FAURE. The Treatment of Tabes Dorsalis and its Prognosis. *Lancet*, June 18, 1904, p. 1717.  
 DONADIEU-LAVIT. Du traitement mercuriel intensif dans le tabes spécifique. Montpellier, 1904.  
 R. T. WILLIAMSON. Hereditary Syphilitic Tabes (Juvenile Tabes). *Rev. Neurol. and Psychiat.*, June 1904, p. 425.  
**Friedreich's Ataxia.**—PIC et BONNAMOUR. Un cas de maladie de Friedreich avec autopsie. *Nouv. Icon. de la Salpêtrière*, mars-avril 1904, p. 126.  
**Combined Scleroses.**—KINNEAR WILSON and CROUZON. A Case of Senile Combined Sclerosis of the Spinal Cord. *Rev. Neurol. and Psychiat.*, June 1904, p. 431.  
**Disseminated Scleroses.**—MARK BERMANN. Ein Beitrag zur Kasuistik der Lehre von der cerebrospinalsklerose. Unilaterales Intentionszittern. *Wien. med. Wchnschr.*, Mai 28, 1904, S. 1005.  
**Syringomyelia.**—W. LANGE. Ein Fall von Syringomyelia. *St. Peterb. med. Wchnschr.*, Mai 15, 1904, S. 214.  
 BORCHARD. Die Bedeutung des Traumas bei der Syringomyelie vom versicherungrechtlichen Standpunkte. *Monatschr. f. Unfallheilk.*, No. 2, 1904.  
 STEINHAUSEN. Syringomyelia als Folge von Rückenmarksverletzung. *Monatschr. f. Unfallheilk.*, Nr. 4, 1904.  
**Aneurism.**—RAYMOND et CESTAN. Un cas d'anévrisme cirsoïde probable de la moelle cervicale. *Rev. Neurol.*, mai 30, 1904, p. 457.  
**Spine.**—FROELICH. La spondylite syphilitique ou mal de Pott syphilitique. *Presse med.*, juin 1904, p. 361.  
 BRISSAUD et GRENET. Un cas de cyphose d'origine articulaire ou musculaire. *Nouv. Icon. de la Salpêtrière*, mars-avril 1904, p. 85.  
 FORESTIER. Déformations rachidiennes: 1. Sciatique avec cyphose et scoliose homologue; guérison et redressement complet; 2. Trois cas de spondylose rhumatismale ankylosante. *Nouv. Icon. de la Salpêtrière*, mars-avril 1904, p. 88.  
**Cytodiagnosis.**—MILIAN. Le liquide céphalo-rachidien. Steinheil, Paris, 1904, 6 fr.  
 MERZBACHER. Ergebnisse der Untersuchung des Liquor cerebrospinalis. *Neurol. Centralbl.*, Juni 16, 1904, S. 548.  
 SIEMERLING. Ueber den Werth der Untersuchungen des Liquor cerebrospinalis für die Diagnose der Nerven- und Geisteskrankheiten. *Berl. klin. Wchnschr.*, Mai 23, 1904, S. 549.  
 WERTHEIMBER. Ueber den diagnostischen und therapeutischen Wert der Lumbalpunktion bei der Meningitis. *Münch. med. Wchnschr.*, Juni 7, 1904, S. 1004.  
 ISADOR H. CORIAT. The Chemical Findings in the Cerebro-Spinal Fluid and Central Nervous System in Various Mental Diseases. *Am. Journ. Insan.*, April 1904, p. 733.  
 NISSEL. Die Bedeutung der Lumbalpunktion für die Psychiatrie. *Centralbl. f. Nerven- u. Psychiat.*, April 15, 1904, S. 225.  
 VALETTE. La Lymphocytose du liquide céphalo-rachidien, élément de diagnostic dans l'hémiplégie syphilitique. Michalon, Paris, 1904, 1 fr. 25.  
 VOULCOFF. Contribution à l'étude du liquide céphalo-rachidien dans la paralysie générale. Thèse. Firmin, Montane et Sicardi, Montpellier, 1904.

## BRAIN AND MENBRANES—

- G. PAGANO. Saggio di localizzazioni cerebellari. *Riv. di Patolog. nerv. e ment.*, Vol. ix., f. 5, 1904, p. 209.

- Meningitis.**—HENRY KOPLIK. The Clinical Features of Cerebro-spinal Meningitis or Cerebro-spinal Fever of the Abdominal Type. *Med. News*, June 4, 1904, p. 1065.
- Hæmorrhage.**—FREDERICK LANGMEAD. On Three Cases of Suprarenal Apoplexy in Children. *Lancet*, May 28, 1904, p. 1496.
- Hemiplegia.**—HENRY FRASER. A Case of Hemiplegia occurring during Whooping Cough and Diphtheria. *Pediatrics*, June 1904, p. 286.
- T. H. WEISENBURG. Uremic Hemiplegia, with Changes in the Nerve Cells of the Brain and Cord, and Recent Primary Degeneration of the Central Motor Tract. *Proc. of the Path. Soc. of Philadelphia*, Feb. 1904.
- CONSTANTIN. L'Hémichorée par lésion organique. Thèse. Firmin, Montane et Sicardi, Montpellier, 1904.
- Cerebral Tumours.**—ED. CORNU. Un cas de tumeur cérébrale à forme psychoparalytique. *Nour. Icon. de la Salpêtrière*, mars-avril 1904, p. 107.
- THEODORE DILLER. Brain Tumor involving the Superior Parietal Convolution: Two Operations for its Removal: Partial Success, with some Relief of Symptoms: Death, Two Years Later: Autopsy. *Am. Journ. Med. Sc.*, March 1904.
- WALTER D. BERRY. Intracranial Psammo-Sarcoma without Paralysis. *Am. Journ. Insan.*, April 1904, p. 787.
- J. RAMSEY HUNT. Congenital Cysts of the Fourth Ventricle. Report of two Cases associated with Tumor of the Optic Thalamus and Crus Cerebri. *Am. Journ. Med. Sc.*, March 1904.
- F. MARCHAND. Über Gehirnsystirzerken. *Samml. Klin. Vortr.*, Mai 1904, S. 186.
- Brain Syphilis.**—PRITCHARD. A Diagnostic Syndrome for Intracranial Syphilis, with Remarks upon Prognosis and Treatment. *Med. Rec.*, May 14, 1904, p. 769.
- M. BERTOLOTTI. Il segno d'Argyll e la meningite sifilitica. *Riv. crit. di clin. med.*, Anno v., Nos. 23 e 24, 1904.
- General Paralysis.**—THEOPHILE KOETSCHET. Progressive Paralyse und Syphilis mit Bertoktsichtigung der in Boenian und Harcegovina gesammelten Erfahrungen. *Wien. med. Wchnschr.*, Juni 11, 1904, S. 1110.
- GREENLEES. The Circulatory Apparatus in General Paralysis of the Insane. *Caledonian Med. Journ.*, June 1904, p. 416.
- WM. A. WHITE. The Early Diagnosis of General Paralysis. *Med. News*, April 9, 1904.
- BUMKE. Zur Pathogenese der paralytischen Anfälle. Zugleich ein Beitrag zur Anatomie der Pyramidenbahn. *Neurol. Centralbl.*, Mai 16, 1904, S. 436.
- PASTUREL. Paralytiques généraux perscutés et dangereux. *Arch. de Neurol.*, juin 1904, p. 456.

#### GENERAL AND FUNCTIONAL DISEASES—

- Chorea.**—HORACE E. MARION. A Many Phased Case of Chorea. *Boston Med. and Sur. Journ.*, April 7, 1904.
- J. J. PUTNAM. A Case of Huntington's Chorea: its Social Aspects. *Boston Med. and Sur. Journ.*, April 7, 1904.
- ISABEL A. BRADLEY. A Case of Chorea Insaniens with Report of Autopsy. *Am. Journ. Insan.*, April 1904, p. 777.
- Epilepsy.**—F. S. PEARCE and L. N. BOSTON. The Blood in Epilepsy: Experiments on Animals. *Am. Journ. Insan.*, April 1904, p. 597.
- L. PIERCE CLARK and THOMAS P. PROUT. Status Epilepticus: A Clinical and Pathological Study in Epilepsy. *Am. Journ. Insan.*, April 1904, p. 645.
- SCHWARZ. Ueber "Heus hystericus." *St Petersburg. med. Wchnschr.*, Mai 22, 1904, S. 226.
- SAVILL. Clinical Lectures on the Pathology of Hysteria. III. Hysterical Motor Disorders. *Clin. Journ.*, June 1, 1904, p. 103.
- TH. DILLER. Severe Case of Major Hysteria (Traumatic Neurosis). *Med. News*, May 14, 1904.
- WM. B. NOYES and RICHARD C. NEWTON. Hysteria and Organic Disease. *Med. News*, April 16, 1904.
- Hysteria.**—DEBOVE. Hystéro-traumatisme à manifestations cardiaques. *Arch. gén. de méd.*, juin 14, 1904, p. 1499.
- Neurasthenia.**—C. L. DANA. The Partial Passing of Neurasthenia. *Boston Med. and Sur. Journ.*, March 24, 1904.
- Exophthalmic Goitre.**—LEROY CRUMMER. Exophthalmic Goitre. *Western Med. Rev.*, March 15, 1904.
- H. SALOMON. Gaswechseluntersuchungen bei Morbus Basedowii und Akromegalie. *Berl. klin. Wchnschr.*, Juni 13, 1904, S. 635.

- ARTHUR W. ROGERS. Treatment of Graves' Disease. *Wisconsin Med. Journ.*, April 1904.  
 E. BOIX. Traitement du goître exophtalmique. *Arch. gén. de méd.*, juin 14, 1904, p. 1504.  
 Myxedema.—WOLSELEY-LEWIS. The Mental State in Myxedema. *Lancet*, April 23, 1904, p. 1117.  
 Adipositas Dolorosa.—SCHWENKENBECHER. Ueber die Adipositas dolorosa. *Deutsches Arch. f. klin. Med.*, Bd. 80, H. 3 u. 4, 1904, S. 317.  
 Mentalhypertrophy.—ROBERT HUTCHISON. A Case of Hemi-Hypertrophy in which the Internal Organs were affected. *Brit. Journ. Child. Dis.*, June 1904, p. 258.  
 Sleeping Sickness.—ROUJAS. La Maladie du sommeil. Thèse. Naud, Paris, 1904.

## ALCOHOL, ETC.—

- STOLL. Alkohol und Kaffee in ihrer Wirkung auf Herzleiden und nervöse Störungen. H. Friedrich, Karlsruhe-Berlin, 1904, M.—50.  
 TOMASINI. Sulla psicosi cocainica. *Manicomio*, Anno xx., No. 1, 1904, p. 67.  
 J. M. BUCHANAN. The Treatment of the Morphia Habit by Hyoscine. *Am. Journ. Insan.*, April 1904, p. 635.

## MENTAL DISEASES—

- KRAEPELIN. Clinical Psychiatry. Authorized Translation, edited by Thos. Johnstone. Baillière, Tindall & Cox, London, 1904, 10s. 6d.  
 Instruction in Psychiatry. Editorial. *Journ. Am. Med. Assoc.*, May 28, 1904, p. 1420.  
 ROBERT JONES. The Development of Insanity in Regard to Civilization. *Am. Journ. Insan.*, April 1904, p. 577.  
 NIESSL. Zu Kronthal's Aufsatz: Nervenselle und Psychose. *Centralbl. f. Nervenhk. u. Psychiat.*, Mai 15, 1904, S. 307.  
 JAMES HENDRIE LLOYD. The Metaphysical Conception of Insanity. *Journ. Nerv. and Ment. Dis.*, June 1904, p. 374.  
 W. K. WALKER. A few Considerations concerning the Mechanism of Developing Mental Disorder. *Med. News*, May 14, 1904, p. 928.  
 J. HEITZ. Nouveaux documents sur les possédés et les malades dans l'art byzantin. *Nouv. Icon. de la Salpêtrière*, mars-avril 1904, p. 159.  
 ESPOSITO. Sulle malattie mentali familiari (cont. e fine). *Manicomio*, Anno xx., No. 1, 1904, p. 1.  
 DEL GRECO. La follia di Re Lear. *Manicomio*, Anno xx., No. 1, 1904, p. 100.  
 LEVI BIANCHINI. Sulla patogenesi del mutacismo (sommissione del linguaggio) nella demenza primitiva. *Manicomio*, Anno xx., No. 1, 1904, p. 116.  
 C. A. DREW. Was he a Paranoiac? *Am. Journ. Insan.*, April 1904, p. 611.  
 W. RUSH DUNTON. Report of a Second Case of Dementia Precox with Autopsy. *Am. Journ. Insan.*, April 1904, p. 761.  
 E. LUGARO. Sulle allucinazioni unilaterali dell' uditio. *Riv. di Patolog. nerv. e ment.*, Vol. ix., f. 5, 1904, p. 228.  
 MARCHAND. Considérations sur l'ampliation des ventricules latéraux dans les maladies mentales. *Journ. de Neurol.*, mai 20, 1904, p. 181.  
 STADELMANN. Das Wesen der Psychose auf Grundlage moderner naturwissenschaftlicher Anschauung. 2 u. 3 Heft. Grund u. Ursache der Psychose. Der Kontrastcharakter. Die Hysterie. Ballhorn & Cramer, Würzburg, 1904, M. 3.  
 BOURDON. Étude sur la stasobasophobie. Le Bigot frères, Lille, 1904.  
 REMOND. Précis des maladies mentales. Rudeval, Paris, 4 fr.  
 FEUILLADE. Antipyrine et Aliénation mentale. Poinat, Lyon, 1904.  
 MASSELOIN. Forme fruste de démence précoce. *Arch. de Neurol.*, juin 1904, p. 438.  
 MASOIN. Organisation d'un service de médecine mentale dans les prisons. *Journ. de Neurol.*, juin 5, 1904, p. 201.  
 SOMMER. Kriminalpsychologie und strafrechtliche Psychopathologie auf naturwissenschaftlicher Grundlage. J. A. Barth, Leipzig, 1904, M. 11.50.  
 DUPRÉ. Définition médico-légale de l'aliéné. Gainche, Paris, 1904.

## CRANIAL NERVES—

- O. LANGE. Ueber einen Fall von traumatischer bitemporaler Hemianopsie mit hemianopischer Pupillenreaktion. *Klin. Monatsbl. f. Augenheilk.*, Mai 1904, S. 419.  
 HOFFMANN. Zur Kasuistik der indirekten Verletzungen des Sehnerven. Pletscher, Tübingen, 1904, M. 6.

CHAS. A. OLIVER. Clinical and Histologic Study of the Ophthalmic Conditions in a case of Cerebellar Neoplasm occurring in a Subject with Renal Disease. *Am. Journ. Med. Sc.*, June 1904.

#### REFLEXES—

THEODORE DILLER. Investigation of Certain of the Reflexes and Sensory Phenomena and the Eye Grounds. *Am. Med.*, April 23, 1904.

#### MISCELLANEOUS SYMPTOMS—

M. E. RUMWELL. Case of Aphasia. *Occidental Med. Times*, March 1904.

URBANTSCHITSCH. Über die von den sensiblen Nerven des Kopfes ausgehenden Schrift- und Sprachstörungen sowie Lähmungen der oberen und unteren Extremitäten. *Deutsche Ztschr. f. Nervenheilk.*, Bd. xxvi., H. 3, 1904, S. 199.

JAMES M. BROWN. A Consideration of Speech Defects. *Journ. Am. Med. Assoc.*, May 28, 1904, p. 1417.

LANGDON BROWN. An Unusual Case of Muscular Atrophy. *Lancet*, June 11, p. 1651.

#### TREATMENT—

**Medical.**—W. BLAIR STEWART. The Use and Abuse of Hypnotics. *Journ. Am. Med. Assoc.*, May 21, 1904, p. 1835.

STEPHANE LEDUC. Traitement des névralgies par l'ion salicylique. *Arch. d'Elect. méd.*, juin 10, 1904, p. 403.

BENI-BARDE. Du traitement hydrothérapique de la neurasthénie. *Journ. des Prat.*, mai 21, 1904, p. 328.

MAURICE DECHAUFFOUR. Traitement préventif des Atrophies Musculaires réflexes et des Ankyloses dans les Lésions traumatiques ou inflammatoires des Articulations. *Thèse de Paris*, Jouve, 1904.

R. D. BAKER. Physiological and Clinical Aspects of Hydrotherapy with its Special Reference to the Treatment of Psychoses. *Med. News*, March 26, 1904.

MARCO LEVI BIANCHINI. Ergothérapie et psychothérapie. *Nouv. Icon. de la Salpêtrière*, mars-avril 1904, p. 136.

WM. C. SEBRING. What shall we do with the Nervous Woman? *Med. Rec.*, April 9, 1904, p. 564.

S. WEIR MITCHELL. Evolution of the Rest Treatment. *Journ. Nerv. and Ment. Dis.*, June 1904, p. 368.

CAMUS et PAGNIEZ. Isolement et Psychothérapie. Traitement de l'hystérie et de la neurasthénie. Pratique de la rééducation morale et physique. F. Alcan, Paris, 1904, 9 fr.

WILHELM. Die modernen Nervenheilstalten. Welchen Nutzen haben sie für Nervöse? Reilinger, Wien, 1904, M.—70.

DENIS COURTADE. Technique du traitement électrique de la névralgie faciale par les courants continus. *Journ. des Prat.*, mai 14, 1904, p. 306.

**Surgical.**—J. C. DA COSTA. The Surgery of Idiocy and Insanity. *Journ. Nerv. and Ment. Dis.*, June 1904, p. 386.

RICHARD LAKE. Removal of the Semicircular Canals in a Case of Unilateral Aural Vertigo. *Lancet*, June 4, 1904, p. 1567.

DONALD ARMOUR. The Surgery of the Gasserian Ganglion. *Practitioner*, June 1904, p. 898.

WM. P. SPRATLING and G. M. GOULD. Non-operative Relief of Eye-strain for the Possible Cure of Epilepsy as Tested in 68 Cases at the Craig Colony. *Am. Med.*, April 9, 1904.

DEROCQUE. Les Résultats éloignés de la transplantation tendineuse dans la paralysie infantile. Lecerf fils, Rouen, 1904.

H. M. W. GRAY. Transplantation of Tendon for Musculo-spiral Paralysis. *Lancet*, May 21, 1904, p. 1416.

MOTONOSUKE GOTO. Dehnungsversuche an gelähmten Muskeln. *Ztschr. j. Biol.*, Bd. 46, H. 1, 1904, S. 38.

MORRIS MANGES. Intraspinal Injection of Lysol Solution in the Treatment of Cerebro-spinal Meningitis; with Report of Three Cases. *Med. News*, May 14, 1904, p. 913.

HARDOUIN et PASQUIER. Observation de fracture du crâne opérée d'urgence à la campagne, dans une salle de ferme. Perte d'environ 15 grammes de substance cérébrale. Ablation de vastes esquilles. Guérison sans troubles cérébraux. *Ann. méd.*, mars 1904, p. 74.

DEVILLIERS. De la nécessité d'une intervention immédiate dans les traumatismes crâniens. *Thèse de Paris*, mars 1904.

CHIPAULT. Chirurgie nerveuse d'urgence. Baillière et fils, Paris, 1904, 1 fr. 50.

WALLACE BLANCHARD. Present Status of the Mechanical Treatment of Spondylitis. *Chicago Med. Recorder*, April 15, 1904.



# **Review**

of

## **Neurology and Psychiatry**

---

### **Original Articles**

#### **OPTIC NEURITIS IN CASES OF INTRACRANIAL TUMOUR, WITH SPECIAL REFERENCE TO THE NEUROGLIAL CHANGES PRESENT.**

By **ROBERT A. FLEMING, M.A., M.D., F.R.C.P. Edin.,**  
Assistant Physician to the Royal Infirmary, Edinburgh ;  
Lecturer on Practice of Medicine, School of the Royal Colleges, Edinburgh.

THE results of the microscopic examination of a number of optic nerves, mostly from cases of intracranial tumour, abscess, and meningitis, appeared to offer a new verification for an old theory with regard to the nature of optic neuritis in cases of intracranial tumour, namely, that a toxin of some special kind is responsible for the condition. Further, they completely corroborate the view that the neuroglia so-called, really consists of two quite distinct elements, neuroglial cells proper, believed to be of epiblastic, and mesoglia cells of mesoblastic origin, and they might be taken as a pathological confirmation of what Ford Robertson (1) has shown by his platinum method, that mesoglia cells are absolutely distinct from neuroglial.

I purpose giving: 1st—A brief account of the theories of optic neuritis as regards its occurrence in cases of intracranial tumour.

2nd—An account of the microscopic appearances described in optic neuritis, and specially the neuroglial changes which I have found in my specimens.

3rd—A note of a few representative cases of optic neuritis with photographic illustrations.

The first theory, von Graefe's, was that venous obstruction from increased intracranial pressure, especially affecting the cavernous sinus, might account for the optic neuritis. Needless to remark, the free anastomosis with facial and orbital veins, renders this theory untenable. Broadbent (2), Manx (3), and others believed that the great pressure of lymph in the inter-sheath space might cause the optic neuritis. While it is true that a post-bulbar distension of the inter-sheath space is not uncommon, it may be slight even in extreme cases of optic neuritis. Schmidt included the lymph spaces, which form such an important means of communication between the inter-sheath space and the lymphatic channels in the nerve and pial sheath.

It is usually pointed out in referring to these three theories, that while a very small tumour may produce the most intense optic neuritis, a very large one may cause none at all.

Leber (4) believes that there is an irritant of some kind in the cerebro-spinal fluid.

Galezowski, Brailey, Edmonds, Juler, and others, hold that the irritant is not in the cerebro-spinal fluid, but in the brain tissue itself, by which it is directly conducted downwards to the optic nerve.

Schweller and Hughlings Jackson suggest that the papillar inflammation is due to a reflex stimulation of the vaso-motor nerves of the disc.

Deutschmann (5) closely associates meningitis with optic neuritis, partly as the result of experiments on rabbits, in which he found that injecting staphylococci or tubercle bacilli into the subarachnoid space produced a more or less definite optic neuritis, and partly because in most cases of optic neuritis due to intracranial tumour, he describes a perineuritis with not infrequently a certain amount of localised meningitis in the neighbourhood of the tumour. Gowers believes that it is a descending neuritis which is present in intracranial tumour cases, and often associated with meningitis; the nuclear increase in the optic nerve and inter-sheath space he believes to be mostly of inflammatory origin.

In connection with this, Barlow (6) and others consider that in tubercular meningitis, when a late stage is reached, definite clinical evidence of optic neuritis is always found, namely, blurring

of the edges of the disc, covering of vessels, and great alteration in relative size of arteries and veins.

I began this research several years ago with the anticipation that I should find the choked disc theory most nearly the correct one, and with the hope that relief obtained by draining away some of the cerebro-spinal fluid might be expected to prove sufficient in most cases to arrest the optic neuritis. I soon realised that choking of the papilla by lymph pressure within the inter-sheath space was by no means the rule, while I was struck by the fact that in a very large proportion of the cases of tubercular or other infective meningitis in which pressure might be expected, there was no microscopic evidences of true optic neuritis, except a slight leucocytic invasion of the periphery of the nerve, and often with no (post-mortem) appearance of papillitis at all.

This paper is based essentially on post-mortem results, and therefore it is desirable to remind the reader that, in examining the optic nerves microscopically, so as to recognise the presence of optic neuritis, some definite pathological alteration must be looked for. Just as in peripheral neuritis of very recent origin, there may be no appreciable microscopic change in the nerve fibres or segmental nuclei, even when a very definite and easily recognisable lesion exists in the nerve-cells, so it is extremely probable that in a recent case of optic neuritis—provided optic neuritis is really parenchymatous—nothing may be seen after death to bear out the ophthalmoscopic and clinical evidence of an early inflammation of the papilla. The changes I am about to describe are so definite when fully developed that they are both unmistakable, and can hardly disappear with the rapidity which clinical experience teaches us the optic neuritis may vanish after the removal of an intracranial tumour.

Brudenell Carter (7) contends that it is absurd to call all cases of swelling of the discs with distension and tortuosity of the retinal veins by the term optic neuritis unless sight is actually failing or affected; and he complains that the term is often mostly loosely used in speaking of cases of intracranial disease. The swelling of the papilla may not interfere with the conductivity of the nerve fibres, and, if so, are we warranted in calling the condition inflammatory? Buzzard believes that patches of what would eventually become sclerosis may develop

anywhere in the visual tract associated with similar patches elsewhere, and that they may disappear. Such opinions as that expressed by Brudenell Carter make it very necessary to inquire what the microscopic changes in optic neuritis really are. It is certainly inflammatory, and the swelling of the papilla is due to inflammatory lymph, with a varying number of cells.

In the inter-sheath space there may be a considerable number of cells, but not necessarily so. The arachnoid often seems unduly thickened and cellular, but only in a very small percentage of my cases do I feel justified in employing the term perineuritis.

The nerve itself certainly shows a nuclear increase, and it is the explanation of this nuclear increase which is the special point I wish to describe. In an early case of optic neuritis, or where optic neuritis has not been marked, no very definite change is present; but where it is very advanced before death, there is a sharp distinction between the two types of cells which used to be called neuroglial. The normal neuroglial cells may well be described in the words of Ford Robertson (8), as "essentially highly branched cells, which vary greatly in size, and in the number and arrangement of their processes. Their nuclei are oval or rounded in form, have a prominent nuclear membrane and chromatic filaments, distributed evenly throughout the nuclear matrix. Their protoplasm, originally large in amount, has become in greater part differentiated into a denser substance, which forms fine fibres." It is beside my purpose to discuss the question as to whether these fibres become distinct from the cell or not; and I need only add that the function of these neuroglial cells is largely that of supporting structures for vessels, nerve fibres, etc. It is very difficult to differentiate them in a normal optic nerve, by the method of fixing and staining I use, from the second type of cells—the mesoglia—but in the optic neuritis of intracranial tumour the distinction is most obvious, and their arrangement in the funiculus of a healthy nerve also becomes more clear. These neuroglial cells undergo the following very definite changes, which are well seen in the micro-photographs shown.

1. They proliferate, and the proliferation as studied in longitudinal sections shows that they run in fairly definite rows in the normal nerve, an arrangement which aids the trabeculae in the support of the optic nerve fibres.

2. The protoplasm of the cell is enormously increased and

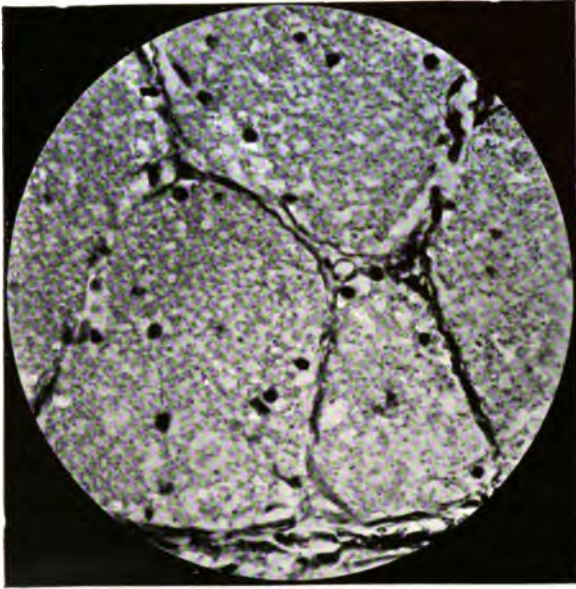


FIG. 1.

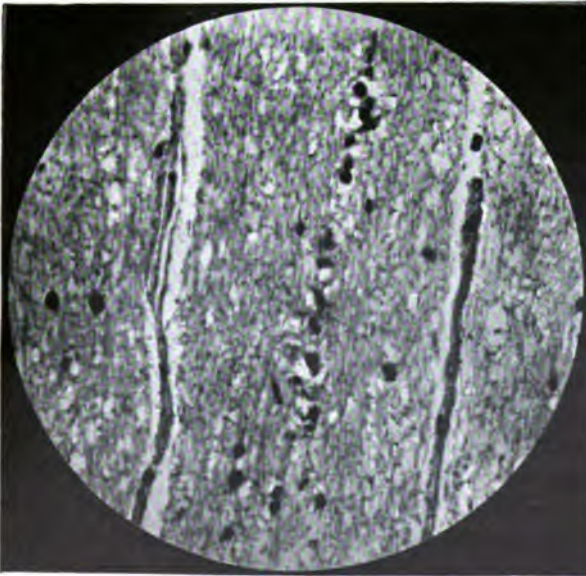


FIG. 2.

Normal optic nerve. Transverse and longitudinal sections, behind entrance of central artery of retina. This specimen was obtained from a man, æt. 26, who died from fracture of the skull. Hamatein and Benzo-purpurin.  $\times 300$ .



the processes become much more visible, extending quite obviously for long distances amongst the nerve fibres.

3. Many of the larger cells have a distinctly granular cytoplasm, and the periphery is often blurred. In some of my cases vacuolation is very marked (see photograph 6).

4. The nuclei of the cells become much larger, show definite karyokinetic figures, and as a whole, stain more faintly than the normal neuroglial nuclei do. The nuclei are usually eccentrically placed, but this is a feature of many, if not most, normal neuroglial cells, and is merely enhanced by the great enlargement of the cells.

These neuroglial changes are best marked near the outer portion of the optic nerve, but in an advanced case they are easily recognisable in almost any part of a transverse section, and they may be well seen all along the optic nerve. So far as my observations go, the neuroglial changes are extremely marked just where the central artery of the retina enters the nerve, although they are also very obvious both peripherally towards the eyeball and upwards as far as the optic foramen, beyond which point I have not as a rule been able to obtain the optic nerves.

The cases which show these changes least markedly were undoubtedly those in which the optic neuritis was not so definite during life and where sight was less affected. The mesoglia cells appear in my sections, both normal and pathological, as dark nuclei with very little surrounding protoplasm and few recognisable processes; they do increase in number but not to the same extent as the neuroglial cells, and they do not show any definite structural change.

There are sometimes leucocytes present in the funiculi, but only where meningitis with a marked and quite exceptional perineuritis is present, and their presence can always be recognised by a careful study of the vessels and lymph spaces because leucocytes are always in excess in or near these vessels. I show two photographs (9 and 10) of an optic nerve, one transverse and one longitudinal, from a case of tubercular meningitis with marked purulent exudation and involvement of the inter-sheath space. If these are compared with the corresponding photographs from a normal optic nerve (1 and 2), it will be seen that there is practically no increase of nucleated cells in the funiculi shown, but in or near the vessels and lymph spaces there are numerous

lymphocitic leucocytes which are in excess in the blood stream, and have also made their way into the lymphatics of the trabeculæ from the inter-sheath space. This is not an isolated case but is the picture common to the optic nerve in tubercular meningitis so far as my experience goes.

There is also described in optic neuritis an increase of fibrous tissue, and in cases of optic atrophy this becomes excessive. The increase is specially obvious in the trabeculæ and pial sheath.

The optic nerve fibres very greatly diminish in number in an advanced case, and their axis cylinders swell up in parts, but whether this change is artificial or not it is hard to say. Photographs 7 and 8 show the contrast between the axis cylinders in a normal nerve, and those in Case II. of optic neuritis. These specimens were stained by Stroebe's method. My impression certainly is that the neuroglial changes are of primary importance and not merely secondary to a parenchymatous neuritis, and in fact there is no definite evidence in my specimens of a parenchymatous lesion in the optic nerve fibres; but even with the series of cases of intracranial tumour now at my disposal, numbering just over forty, I cannot at present offer conclusive proof of this suggestion with regard to the absence of a parenchymatous lesion in the nerve fibres.

One is familiar with the statement that optic neuritis, although nearly always bilateral in cases of intracranial tumour, is generally more marked on one side than the other, and not infrequently upon the side on which the tumour or tumours are situated. Microscopically this distinction is not so obvious, and only in Case I. was there much difference between the two eyes.

It is difficult to say where the inflammation unquestionably present begins, whether at the papilla and ascending from there, or descending from the brain. There is no evidence that it is an ascending neuritis, a hypothesis largely based upon the assumption that the choked disc theory is the correct explanation of optic neuritis. There is generally some post-bulbar swelling due to a greater or less degree of distension of the inter-sheath space, but there is no definite constriction of the vessels at the lamina cribrosa. I believe the distension of veins and narrowing of arteries are due in an early neuritis to the pressure of inflammatory lymph inside a pial sheath which cannot stretch,



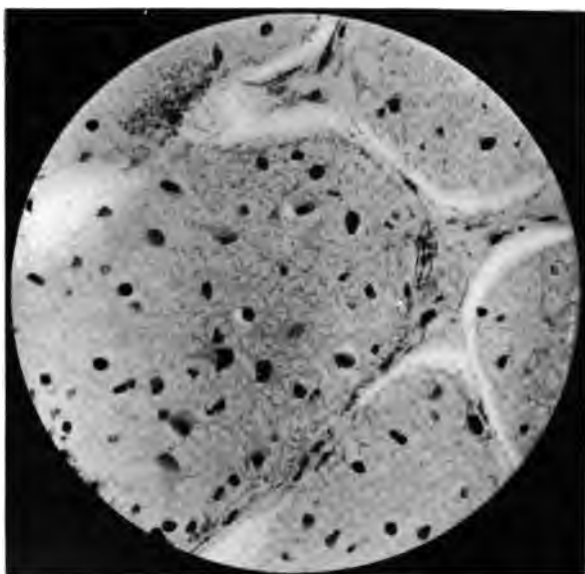


FIG. 3.

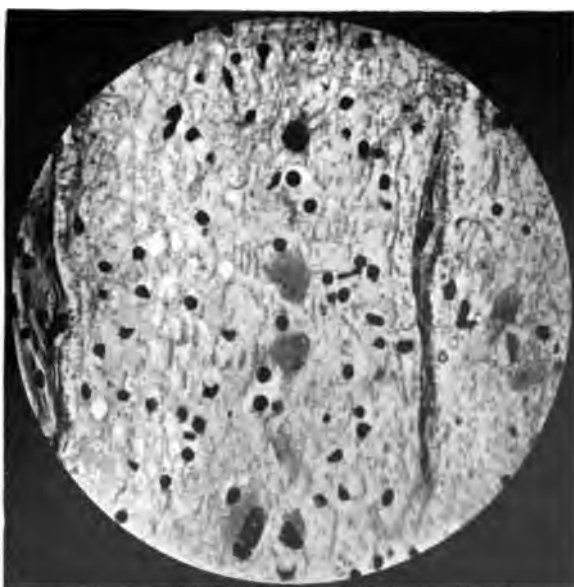


FIG. 4.

Transverse and longitudinal sections from Case 1 referred to in text. They are taken from the optic nerve immediately behind entrance of central artery. Haematein and Benzopurpurin.  $\times 300$ .



and in later stages the neuroglial changes and thickening of the trabeculae may account in part at least for the condition.

I examined a limited number of cases of optic neuritis not due to intracranial tumour, and so far as my experience goes, the extent of neuroglial change is very slight and in no degree comparable to that met with in intracranial tumours, and, if present, is confined to those cases which have, as I believe, a special and perhaps allied toxic element present as the etiological cause of the optic neuritis.

I must refer, however, to the changes which are recognised as occurring in neuroglial cells in repair of brain tissue, as the result of irritation, and in disease. Repair of brain tissue is largely due to the proliferating neuroglial cells, and as the result of certain irritants these cells hypertrophy or swell up. According to Goodall (9), they may do so in 28 hours, and they multiply by division, the nuclei showing very definite karyokinesis. They are generally described as staining deeply, both nucleus and cell apparently acquiring a greater affinity for certain staining reagents, although in my specimens of optic neuritis they stain much more faintly, and the nuclei are often almost ghost-like. The further description of the effects of irritants upon these cells is specially interesting. They develop new dendritic processes, and when the action of the irritant is passing off the protoplasm partially forms fibrils, the cells shrink, eventually reaching their original size, and the tissue is now more or less sclerosed. Ford Robertson points out that neuroglial sclerosis as a process of repair causes little contraction of the area involved. While this may be generally true, in many optic neuritis cases the huge neuroglial cells press on and appear to involve numerous optic nerve fibres.

The neuroglial cells proliferate and hypertrophy in general paralysis and in some other forms of insanity, and also where a patch of cerebral softening has occurred from interference with the blood supply similar changes occur locally. In my specimens the hypertrophy is very great, greater certainly than in any patch of sclerosis, and the blurred outline of the neuroglial cells is very evident while vacuolation is present in advanced cases. Similar blurring has been described by Ford Robertson in certain cases of general paralysis and in experimental uræmia by Sacerdotti and Ottolenghi (10).

It will make my contention clear if I state that in optic atrophy dependent on conditions other than intracranial tumour, as, for example, after enucleation of the eyeball, these advanced neuroglial changes were conspicuous by their absence. It may be urged that they had been present and had disappeared, but I do not think so, and while I do not deny that a degree of hypertrophy was probably present, it cannot possibly have been so pronounced. Perhaps the best proof of this is a somewhat indirect one. Several of my cases of optic neuritis had reached the stage of very definite atrophy before death, and yet in them the neuroglial changes were still as marked as in much more recent cases. This implies that optic atrophy secondary to intracranial tumour differs from optic atrophy due to other causes; and even if it were merely a difference of degree of hypertrophy of the neuroglial cells, which I doubt, it is at least exceedingly marked.

The mesoglia cells may proliferate under the effects of an irritant, but do not show any increase of protoplasm or processes. In my specimens of optic neuritis due to cerebral tumour there seems little doubt that they increase in number.

I propose now offering a few notes of the cases of optic neuritis explanatory of the photographs shown, and before doing so to indicate the methods employed in examining the nerves.

All the eyes were fixed in Müller's Fluid and cut in paraffin, mineral naphtha being employed for the purpose of saturation. The staining methods were hæmatein and benzo-purpurin, and Stroebe's method, anilin blue and saffranin, the latter method for the axis cylinders in longitudinal sections. Benzo-purpurin stains the axis cylinders a rich brown, distinct in shade from fibrous tissue, while Stroebe's method, which may sometimes be found disappointing, was only occasionally used. The uniformity of methods of fixing and staining was adopted so as to arrive at a definite and constant basis for comparison between specimens. Each eye was cut with an optic nerve of 1-1½ inches in length, and both longitudinal and transverse sections were studied behind and in front of the entrance of the central artery of the retina, and the papilla was cut longitudinally. Müller's Fluid is unsatisfactory for fixing the papillary swelling in optic neuritis, especially as objection is taken in this country to the removal of the eyes entire, but the changes to which I have

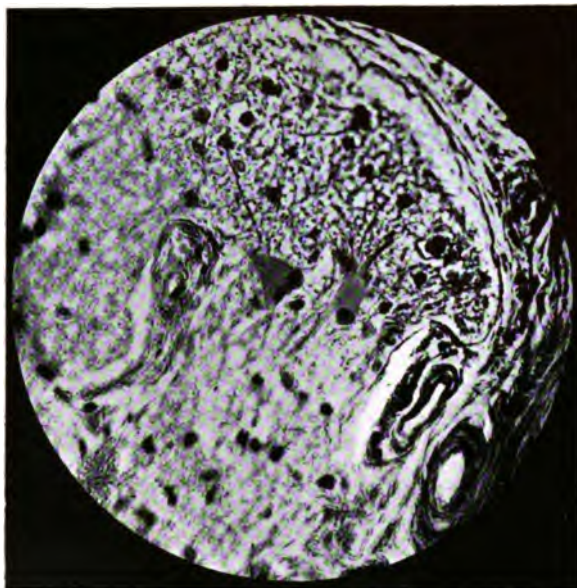


FIG. 5.



FIG. 6.

Sections from Case 2 described in text. Fig. 5 is slightly out of focus in part of the photograph. The sections correspond in position in optic nerve to preceding case. Note in Fig. 5, which is a longitudinal section, the very definite double nucleus in one of the hypertrophied neuroglial cells. In Fig. 6 the vacuolation of the protoplasm is especially clear in one neuroglial cell. Hæmatein and Benzo-purpurin.  $\times 300$ .



specially devoted attention are those in the optic nerve behind the eyeball. The whole optic nerve was cut so that the different parts might be compared. The only hint about staining, should anyone desire to employ these methods, is to keep the sections in both the hæmatein and the anilin blue for a very long time, for the former fully thirty minutes and for the latter two to three days.

I show photographs from two cases of intracranial tumours.

CASE I. was a female, æt. 44, who died of a gumma involving the dura over a wide area and infiltrating, or, more correctly, pressing on the brain. The left occipital lobe was specially involved. There were marked optic neuritis for the last two months of life with commencing failure of vision. The neuroglial changes are well seen in photographs 3 and 4, and the definite sharp black nuclei of the mesoglia cells can be easily distinguished from the hypertrophied and proliferated neuroglia. Comparing photographs 3 with 1, there seems little doubt that the mesoglia cells as well as the neuroglia are increased in number.

CASE II. Male, æt. 40, died of a cerebellar tumour—a glioma situated in the middle lobe of the cerebellum. When death occurred, marked optic atrophy was present in both eyes and sight was almost entirely lost. Loss of sight was of two months' duration. The photographs 5 and 6 are taken from this case, and show the advanced changes in the neuroglial cells; and in 6, which is the longitudinal section, the vacuolation of one neuroglial cell is very clearly seen. Photograph 8 is also from this case and should be compared with 7. In neither of these cases was there much post-bulbar swelling, and there was practically no perineuritis. In Case I. the right eye was rather more severely affected than the left, and in both eyes there were several small recent flame-shaped hæmorrhages in or near disc, together with the remains of a few older ones.

Photographs 9 and 10 are, as already stated, from an optic nerve of a case of tubercular meningitis, and they show that the increase of nuclei is due largely to a great invasion of lymphocytes by blood-vessels and lymph channels, while the rest of the nerve shows no more nuclei than are to be found in photographs 1 and 2, which may be taken as representing photographs of a normal optic nerve.

I might have added a synopsis of my other cases of optic neuritis, but I should like to finish working up the optic nerves from several cases of intracranial tumour in which no optic neuritis is present clinically, and which are not yet quite finished.

The theory which appears to me to offer the most likely explanation of the occurrence of optic neuritis in intracranial tumour is that the tumour, either in consequence of its presence at all, or its position in a special region of the brain, causes a toxic change in the optic nerves. I certainly incline to the view that the cerebro-spinal fluid is the agent which conveys the toxin, and this because the neuroglial cell changes are best marked near the pial sheath, and certainly bear some relationship to the position of the lymph channels.

There is a slighter form of the neuroglial change in albuminuric papillitis, but it is a feeble imitation of what obtains in intracranial tumours.

A very large percentage of my cases happen to be cerebellar, and without exception they all show optic neuritis, while in a case of very large gliomatous tumour of the cerebrum no optic neuritis is present at all. This is common knowledge, and it certainly increases the difficulty of explaining the activity of the supposed toxin in some cases and its inactivity in others.

Perhaps out of the wealth of material I now possess and for which I am indebted to many of my senior colleagues in the Royal Infirmary, Edinburgh, something may be learnt about this intricate point.

I must acknowledge the kindness of the pathologists at the Infirmary who were on many occasions good enough to secure valuable optic nerves for me, and also to Mr Richard Muir for his most excellent photographic reproductions of my slides.

#### REFERENCES.

1. *Journal of Mental Science*, 1900, p. 200.
2. *Trans. Oph. Soc.*, vol. i. p. 108.
3. *Deutsch. Arch. f. klin. Med.*, vol. ix., 1871.
4. *Internat. Med. Congress*, London, 1881.
5. "Ueber Neuritis Optica," Jena, 1887.
6. Clifford Allbutt's "System of Medicine," vol. vii. p. 477, 1899.
7. Clifford Allbutt's "System of Medicine," vol. vi. p. 840.
8. "A Textbook of Pathology of Mental Diseases," 1900, p. 175.
9. *Journ. of Path. and Bacteriol.*, 1894, vol. ii. p. 395.
10. *Riv. di patol. nerv. e ment.*, 1897, f. 1.



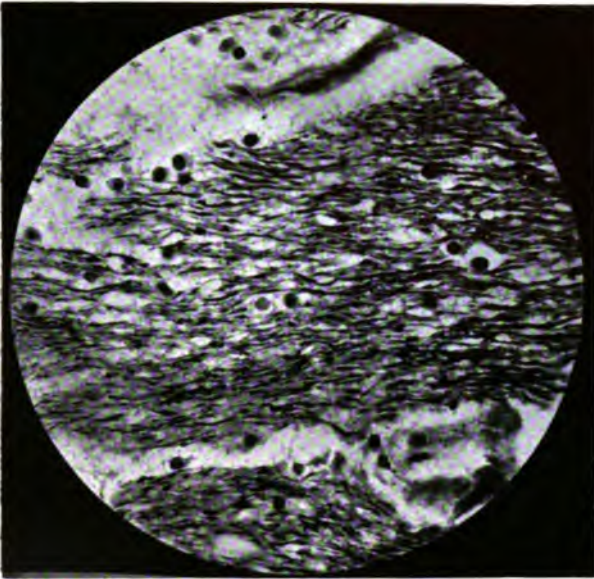


FIG. 7.

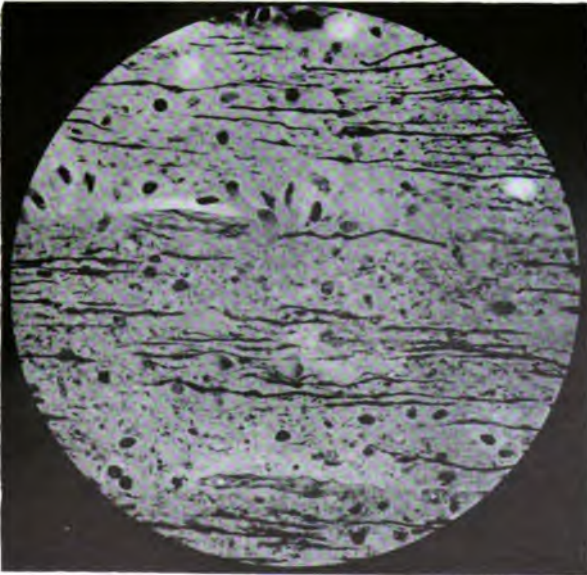


FIG. 8.

These sections show the axis cylinders stained with anilin blue by Stroebe's method. Fig. 7 is from a normal optic nerve, the same case as 1 and 2. Fig. 8 is from Case 2, and there is some distinct localised swelling of the axis cylinder—but occasional swellings are also seen in presumably healthy nerve fibres in Fig. 7.  $\times 300$ .



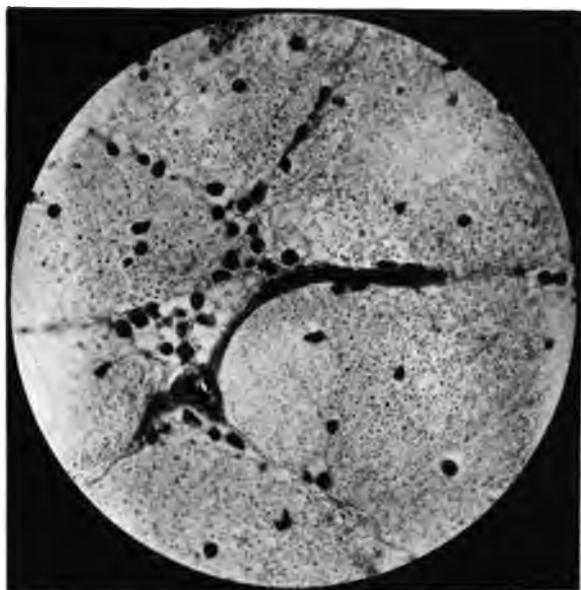


FIG. 9.



FIG. 10.

Transverse and longitudinal sections from the optic nerve of a case of tubercular meningitis in a male, *æ*t. 18. The nuclear increase is evidently lymphocytic, and is entirely confined to the vessels and lymph spaces, as described in the text. Compare carefully with Figs. 1 and 2. Hæmatein and Benzo-purpurin.  $\times 300$ .



**FUNCTIONAL ASTEREOGNOSIS.**

By GEORGE E. RENNIE, M.D., M.R.C.P.Lond.,

Assistant Physician to the Royal Prince Alfred Hospital, and  
Tutor in Medicine, University of Sydney.

It is now well recognised that loss of the stereognostic sense may result from peripheral or central disease. I have shown<sup>1</sup> that in some cases of tabes dorsalis it occurs as a well-marked symptom, and that, in all probability, in this disease it depends upon a loss of conduction of sensory impressions from the periphery; in other words, a loss of some of those sensory impressions such as the spacing, localising, posture senses, the sum total of which appears to be necessary for the accurate perception of the nature and qualities of an object held in the hand with the eyes closed. A condition of astereognosis has also been proved to exist in cases reported by Williamson, Dercum, C. K. Mills and others, where injury has affected the superior parietal lobe.

Now reasoning, *a priori*, it seems not improbable that the same sensory loss might appear as a symptom in purely functional disease, just as we find hemi-anæsthesia, hemi-analgesia, etc., as symptoms in this kind of nerve disease. There seems no valid reason to suppose that "functional degradation of the nerve centres," to use the language of Charlton Bastian, may not affect the superior parietal lobe and result in defective power of forming a correct judgment, even when there may be only a partial peripheral loss of conduction of sensory impressions.

The following case appears to me to be one of this nature, and seems of sufficient interest to warrant publication.

A single man, aged 29 years, a shorthand writer and typist, was sent to me by his family doctor, complaining of some disturbance of sensation in his right hand and left arm. He described this subjective sensation "as if the nerves were going round," and "as if the fingers were being stretched." This condition had lasted seven weeks when I first saw him.

His family history was unimportant, except that he was an only son, and one only sister had died in infancy. There was no special hereditary factor in his family history. He was a native of Manchester, England, and had been in New South Wales for

<sup>1</sup> *British Medical Journal*, Feb. 7, 1903.

twenty-two years. He had never been robust, and had had several illnesses of a mild character; he had smoked and drunk moderately, and had had no venereal disease. He had been a violinist, but had not played for more than six to nine months before the onset of his present condition; he appears never to have suffered from violinist's cramp.

For the six months preceding his illness he had been engaged in very hard work, he had spent long hours at shorthand reporting in a law court, and had been engaged for a long time every evening transcribing and typewriting. He used to feel generally tired, but did not experience any special tiredness in his hands or arms till about seven weeks previous to my first seeing him. At that time he was working at night, and while at work he felt the whole of the left side of his body become cold and numb; he described the sensation as if his clothes were all wet and sticking to his side. At the same time his left hand seemed to stiffen so that he could not use it. It was not a general loss of power of the left hand, but only a difficulty in performing fine movements with his fingers. He had had no preceding headache or pain of any kind about his head, and at the time of the attack his consciousness was not disturbed in the smallest degree. He had no feeling of vertigo, and in the course of a week or two the general numbness of his left side disappeared and all the trouble seemed to centre in his left arm and hand. He was of course quite unable to continue his work, as he could not grasp anything firmly with his fingers.

On examination I found him to be a sparely-built man, rather anæmic, of a somewhat neurotic type. He had no headache, but complained of a certain amount of dazed feeling in his head. His sleep was disturbed; his mental faculties were intact; his cranial nerves showed no evidence of disturbance of function; there was no facial anæsthesia.

On examination of the left hand I found that he could perform all movements, but the movements were somewhat clumsily executed, not from loss of power, but from loss of sensation. There was a relative but not absolute anæsthesia and analgesia of the left hand; this was not sharply limited as in the glove type of functional anæsthesia, but gradually merged into perfect tactile and painful sensation a short distance above the wrist. He could readily recognise that an object was in contact with

his hand, and could recognise a sharp object such as a pin as distinct from the finger tip. His muscular sense and sense of posture were defective; with his eyes closed he could not tell the position of his fingers accurately, and could not bring the tip of the forefinger to the tip of the nose with accuracy. His spacing sense was defective; his localising sense was also defective. He did not recognise when pressure was being exerted upon any part of his hand placed palm upwards upon his knee; but he could recognise difference in weights placed upon his hand when held out from the body. He was quite unable to recognise the nature of any object placed in his left hand if his eyes were kept closed. His reflexes were all normal and equal on both sides of the body; he could walk about quite well without any feeling of tiredness, and had no hemiplegic gait; he had no sphincter trouble. His digestive system was normal; his urine was faintly alkaline, contained no albumen or sugar, but showed phosphates on heating. His circulatory system showed nothing abnormal. There was no tenderness on percussion of the cranium or spine, and no tenderness of any nerve trunks.

Six weeks later he had much improved; his left hand grasp was much better, and his astereognosis had practically disappeared, as he could now recognise a threepenny piece, a knife, a pencil, etc., almost at once.

Two months later he was much better; but while his stereognostic sense appeared almost normal, he complained of a sensation in the left hand as if it were covered by a net. He still had some defect in the spacing sense and also in the posture sense, as he could not place the fingers of his right hand in the same position as those of his left with his eyes closed.

When seen seven months later he still had the sensation of the left hand being covered with a net. He had now recovered his spacing, pressure, and posture senses, and could play the violin with his eyes closed without any effort. For several months past he had been living an outdoor life, being engaged in poultry farming, and his general health had much improved.

It is obvious that the astereognosis in this case must have been due to some cerebral condition; in other words, it was not due to defective conduction of sensory impressions, but rather to defective co-ordination of those peripheral sensations. There was absolutely no tenderness of the nerve trunks, the condition was

unilateral, and there was no etiological factor present which would determine a peripheral neuritis or a peripheral neurone degeneration. It is of much interest to note, therefore, that although the special sensations, such as spacing, pressure and posture were defective, yet the sensations of pain and contact were retained. It would appear as if the loss of these special sensations were due to a central defect rather than to a defect in the peripheral receptive or conducting mechanisms, and the only question which remains open for discussion is, whether he had a gross lesion in the sensory pathway in the brain, or merely a condition of defective functional activity of the superior parietal cortex. I incline decidedly to the latter view. For gross lesions, such as hæmorrhage, thrombosis and softening, affecting the sensory pathway, or destroying the grey matter of the superior parietal cortex or the subjacent white matter, would surely have produced a more permanent condition of hemi-anæsthesia.

Further, the entire absence of any cardio-vascular changes which would occasion a vascular lesion, and the absence of any disturbance of consciousness at the onset of affection, are strong points against the hypothesis of a gross vascular lesion. In favour of the view I have adopted, we have a man with a weak constitution, enfeebled by several illnesses, overstrained both mentally and physically, and making a practically complete recovery under a line of treatment adapted to improve his general health. I submit that the evidence is all in favour of this case being an instance of purely functional astereognosia.

---

## ON THE SEGMENTATION OF THE INTERMEDIO-LATERAL TRACT.

### Preliminary Communication.

By ALEXANDER BRUCE, M.D., F.R.C.P.E., and  
C. MACFIE CAMPBELL, M.D.

ONE of the observers in studying the grouping of the motor cells in the cervical enlargement was struck by the appearance in the first dorsal segment of the cord of a segmental grouping of the cells of the intermedio-lateral tract. To ascertain whether this



was the normal arrangement of the cells of this tract the first dorsal segment obtained from another cord was examined and a similar grouping of the cells was observed.

The present work was undertaken to determine whether the segmentation of the intermedio-lateral tract was continued throughout the whole dorsal region of the cord. For this purpose a normal cord was cut into serial sections of uniform thickness from the eighth cervical to the third lumbar segment inclusive. The sections were stained with Unna's polychrome methylene blue and the position and number of cells were noted in each section.

The main result was to establish the fact that a definite segmentation of the intermedio-lateral tract exists throughout the whole length of the dorsal region of the cord. The intermedio-lateral tract of the dorsal region was found not to extend higher than the eighth cervical or lower than the third lumbar segment. (The scope of the research did not include examination of the higher cervical or lower sacral regions.)

The position of the tract varies according to the level. In the first dorsal segment it lies mainly behind the post-postero-lateral group of motor cells, occupying the margin of the grey matter and sometimes projecting actually into the lateral limiting layer. Occasionally in the lower part of the first and upper part of the second dorsal segment small groups of the large motor cells were surrounded by the cells of the intermedio-lateral tract. At a lower level the tract is situated in the region of the lateral horn, of which it occupies sometimes the anterior border, sometimes the posterior border, and sometimes the whole triangular area.

The cells of the intermedio-lateral tract form a group sometimes compact, sometimes more scattered. Where the group was compact, there was no difficulty in identifying it: where, on the other hand, the cells were more scattered, identification of the cells belonging to the group was more difficult, and it was sometimes impossible to separate them from other cells, especially from the numerous cells in the neighbourhood of Clarke's column. No confusion was possible with the large motor cells occasionally included in the tract in the first and second dorsal segments. At several levels posterior to the intermedio-lateral tract there was noted a well-marked group of cells of similar appearance to those forming the tract; this group was found occasionally to run up to and blend with the intermedio-lateral tract, so that it was impossible to separate them: in other places this posterior group was separated by such a considerable interval from the intermedio-lateral tract that it was difficult to look upon the two groups as merely parts of one large group.

When studied in its entirety, the intermedio-lateral tract was found to consist of a longitudinal chain of segments, separated by

intervals of greater or less extent, in which few or no cells of the group appear. The number of segments of the tract corresponding to a root-division of the dorsal cord varied from division to division. These segments were found as a rule to begin gradually, rise to a maximum number of cells, and then decline. From C8 to D4 the segments of the tract were separated by considerable intervals where there was a complete absence of cells of the tract. From D4 to L1 the segments were separated by intervals in which there was usually some scanty representation of the tract.

The form of the segments of the tract is not the same at all levels. In D1, D2, D3 the segments were, as a rule, short and compact, with a large number of cells at the maximum of the segment: and in these divisions of the cord the growth of the segment was much more abrupt than at lower levels, and between the segments were intervals free from the cells of this group. D4 showed a gradual transition to the condition observed in the succeeding divisions of the cord, where the tract was composed of a series of long segments connected by intervals not quite cell-free. In D7 to D10 the individual segments were longer, and in no section were there so many cells as at certain levels of D2 to D4. The greatest number of cells found in any segment at its maximum from D7 to D10 was about thirty, which number was reached only three or four times, while from D1 to D5 a maximum of over fifty was reached about twelve times.

In D10 the segments began to be richer in cells, the average number per section being high, the maximum being about fifty, and the minimum never coming down to zero. In D11 the cell number was less and the segmentation was more regular than in D10, where the tract was so considerable. In L1, L2, the segments were short and separated by cell-free intervals, and in L3 the tract was found to disappear.

The authors hope to make the detailed results of their investigation the subject of a future communication.

---

## Abstracts

### PHYSIOLOGY.

**THE TRUE MOTOR CENTRES.** A. ADAMKIEWICZ, *Neurol. Centralbl.*, (253) 1904, No. 12, p. 546.

As the result of experiments which the author has been conducting since 1900, he comes to the conclusion that the true centres for

voluntary movements lie in the cerebellum, and not, as is generally held, in the cerebral cortex. He has been able to locate these centres definitely. They are situated on the same side of the cerebellum as the muscle groups which they represent. There is a head centre, a trunk centre, and centres for the extremities. The musculature of the extremities has a threefold representation; each anterior and each posterior extremity has its own special centre; the two anterior extremities have a common centre and so have the two posterior extremities, and all four limbs together have a common centre. Thus, the four extremities are represented in the cerebellum by seven motor centres in all.

This is merely a preliminary note. The complete research will be published shortly, in which full details as to methods and results will be given, and its appearance will be looked forward to with great interest by all neurologists.

SUTHERLAND SIMPSON.

**ESSAY ON CEREBELLAR LOCALISATION.** C. PAGANO, *Riv. di* (254) *Patolog. nerv. e ment.*, May 1904, p. 209.

In this paper Pagano relates a further series of experiments on the cerebellum, in continuation of a previous paper published in 1902. The results to which he now comes are not entirely in harmony with his own previous work.

As before, he stimulated the cerebellum in dogs by means of a solution of curara. No preliminary narcotic or anæsthetic was employed.

He claims to have identified various motor centres in the cerebellum. Thus there is a small area at the junction of the lateral lobe with the upper surface of the vermis whose stimulation produces a posture of retraction and adduction of the homolateral upper limb. This is the result of a tonic contraction of the muscles, but the posture is capable of intermission when a voluntary movement has to be performed with that limb. Another area was identified on the posterior surface of the lateral lobe for the homolateral lower limb. Stimulation of the back of the vermis causes an irresistible tendency to fall backwards, whilst stimulation of the anterior end of the vermis causes the animal to press its head forcibly downwards to the ground. The extreme anterior tip of the middle lobe, when stimulated, produces intense psychical excitement, with howls of rage and terror, passing on into general epileptiform fits followed by collapse and death, the latter generally within four hours, sometimes within one hour.

All the above results are produced by subcortical injections of curara. If the curara be injected simply into the arachnoid space,

i.e. on to the surface of the cerebellum, the result is merely somnolence and disordered voluntary movements. The animal appears giddy and exhibits rapid but partial rotatory movements around its own long axis, alternately to each side, with vomiting and salivation.

In contradiction to his former paper, Pagano now states that the cerebellar movements can be produced even after extirpation of the cerebral motor centres. He therefore holds that the influence of the cerebellum on the cord is a direct one, and not, as formerly stated by him, through the intermediation of the contralateral cerebral cortex.

PURVES STEWART.

**REGENERATION OF NERVES.** F. W. MOTT, W. D. HALLIBURTON, (255) and ARTHUR EDMUNDS, *Proc. Physiol. Soc.*, March 19, 1904.

THE report is a preliminary communication of recent work done by the authors on monkeys and cats. They take the view that in the regeneration of a nerve fibre the neurilemma cells play an active part by clearing away degeneration products and preparing a path for the new axis cylinder, which has an exclusively central origin. The possibility of formation of axis cylinders by the neurilemma cells is discussed and denied, and the clinical observations of early return of sensation after suture of a nerve are not considered trustworthy, owing to certain possible fallacies. These fallacies have been avoided as far as possible in the authors' experiments. In cats the nerves were divided in two places by two separate incisions, and the intervening portions pulled out; in some animals the cut ends were then placed in closed drainage tubes to prevent any possibility of reunion. After 100-150 days the animals were anaesthetised and the nerves tested by strong faradic currents. In no case was there any response in the nerve peripheral to the lesion, and the wasted muscles had also largely lost their power of response. Microscopically there were no signs of regeneration.

In another experiment a nerve was cut and sutured and allowed to regenerate; then a portion of the regenerated nerve was excised, and found to consist of new fibres with delicate medullary sheaths. After 10 days the animal was killed and the nerve examined. No degeneration was present above the second lesion, but marked Wallerian degeneration below it. This the authors consider a further argument in favour of the central origin of the axis cylinders.

Other experiments were done in which the effect of voluntary and reflex stimulation of the regenerating nerve was avoided as much as possible by division of posterior nerve roots, and extirpation of the Rolandic area governing the divided nerve. Similar

nerves on either side were divided, sutured and allowed to regenerate. After 60-70 days the animal was killed and the two regenerating nerves compared. Early experiments of this kind showed a delay in regeneration of the nerve of the side which had been previously operated on, but later experiments showed no such differences.

Further experiments with division of posterior nerve roots and semisection of the cord combined are in progress.

PERCY T. HERRING.

#### THE QUESTION OF REGENERATION IN THE SPINAL CORD.

(256) BIKELES, *Neurolog Centralbl.*, June 16, 1904, p. 559.

IN a previous number of the *Neurologisches Centralblatt* (1903, No. 6), Bikeles, who had repeated Kahler's experiments, came to the same conclusion as the latter writer, that complete regeneration is possible in the extra-medullary part of a posterior spinal root, whilst the intra-medullary portion is incapable of "restitutio ad integrum," even under the most favourable conditions. He differed, however, from Kahler in maintaining the existence of "undoubted regenerative processes" in the cord itself, not only on account of the presence of fine nerve-fibres in the cord, possibly endogenous in origin, but chiefly from a consideration of their appearance in longitudinal sections. In a case of rupture of the cord in man from trauma, whilst all other nerve-fibres were absent, a number of delicate fibres of irregular course were seen in the cord directly continuous with the regenerated fibres of the proximal portion of the posterior root. Though the patient survived for ten months after the accident, these regenerated fibres were evidently of much more recent date. Bikeles proceeds to discuss a case recorded by Spiller and Frazier in which the posterior roots were divided in a dog and the animal was kept alive for ten months afterwards. In this instance there was no evidence of any regeneration on the proximal side, either extra- or intra-medullary. This they ascribe to a difference in biological importance. Bikeles would rather attribute it to extrinsic causes, such as deficiency of conducting paths, increased tendency to cicatrization, etc.

PURVES STEWART.

ON THE PHYSIOLOGY OF THE KNEE-JERK. By SCHEVEN (of (257) Rostock), *Centralbl. f. Nervenl. u. Psych.*, June 13, 1904, p. 402.

ON the basis of experiments on rabbits in which the reaction-time of the knee-jerk and the effect of the summation of stimuli were noted, Scheven concludes that the knee-jerk is a true reflex.

C. MACFIE CAMPBELL.

**THE FUNCTIONAL DUALITY OF MUSCLE.** J. JOTZKO, *Journ.* (258) *de Neurol.*, No. 12, 1904, p. 221.

JOTZKO disputes the generally accepted view that a simple twitch is the sole basis of all motor response of a muscle to stimuli; he claims that there exist in muscle fibres two elements, functionally different, and possessed of unequal excitability. Jotzko points out that these facts were observed by Schiff 45 years ago, but that little attention appears to have been paid to his statements. Recent work has confirmed the observations of Schiff, but has also given them a wider and somewhat different interpretation. Schiff, then, described the quick twitch obtained from a fresh muscle as a neuro-muscular contraction due to the excitement of the nerve endings; the slower contraction of a dying muscle (the more sensitive nerve endings being already dead) he called idio-muscular, and this he regarded as due to the direct stimulation of the muscle fibres themselves. Anything which tended to weaken the nerves (anæmia, certain poisons, exhaustion) favoured the appearance of the idio-muscular contraction. Intermediate contractions could also be obtained on the application of the galvanic current when a neuro-muscular contraction occurred on closure of the circuit, but an idiomuscular one lasted during the whole time of flow of the current. Schiff's theory, however, has not found favour as opposed to that of Claude Bernard. The peculiar contraction met with in muscles poisoned with veratrine has been much discussed in this relation; Bezold and Hirt regarded it as a true tetanus following a single stimulation; Fische ascribed the primary contraction to nervous influence, and the prolongation of this to a direct stimulus of the muscle fibre; Bredirman advanced the theory that all muscles contained both pale and red fibres, the former giving the short, the latter the prolonged contraction. Jotzko regards the theory of Bottazzi as best explaining the difficulties; this author advances the hypothesis that muscle consists of two contractile substances, a fibrillar (anisotropic) and a protoplasmic (sarco-plasmic). This is the modern statement of the theory of Schiff; as in the older view the muscle is regarded as equally excitable, directly and indirectly, but according as the stimulus affects the fibrillar or the protoplasmic part a rapid or a slow contraction follows. The contractility of sarcoplasm has not yet been demonstrated directly, but Jotzko holds that this is the only theory capable of explaining the facts of muscular contraction as at present known. Passing to the results of his own work, Jotzko points out that he was able to separate the contraction of a veratrinised muscle into two parts, the initial twitch being followed after complete relaxation by a slow prolonged contraction, the

former being the fibrillar, the latter the protoplasmic response to the stimulus. There is, in fact, a latent period for the sarcoplasmic as for the fibrillar contraction, but this is infinitely more prolonged; the latent period varies according to the functional condition of the muscle; if this is excitable, the protoplasmic contraction occurs so quickly that the graphic record shows an unbroken curve; if, however, the muscle is fatigued, a distinct break occurs between the two contractions. Veratrine is a type of sarcoplasmic excitants; others are potassium carbonate, ammonia, and various anaesthetics. The galvanic current affects both the contractile structures of muscle; the twitch on opening and closing the circuit is the fibrillar response; the more prolonged contraction during the time of flow (galvanotonic contraction, Wandt) is due to the activity of the sarcoplasm. The sarcoplasm is less excitable than the differentiated protoplasm; it requires a larger and more severe stimulus to excite it.

The reaction of degeneration of muscle is of interest in the light of this new theory; after section of its motor nerve a muscle tends to return to an embryonic condition, the fibrillar substance (myoplasm) diminishes, the protoplasmic substance (sarcoplasm) increases in amount. The reaction of degeneration is, then, the normal response of the sarcoplasm; in other words, no reaction occurs to the short stimulus of faradic electricity, but the galvanic current excites the slow prolonged contraction typical of sarcoplasm.

With regard to the polar effects of the constant current, Jotzko points out that a cathodal contraction on closure is only met with in striped muscle, not in unstriped nor in degenerated striped muscle; these show closing contraction at the anode only; so that a new law of polar contraction may be formulated as follows:—the polar effects of the constant current on muscle are due to the results of the excitability of two different contractile elements, the cathodal closing contraction being due to the fibrillar substance, the anodal opening contraction to the sarcoplasm. There are two forms of contraction possible in living muscle, tetanus and tonus. Tetanus consists of a fusion of a number of rapidly occurring twitches; it depends on the activity of the fibrillar substance, and, owing to the exhaustion it induces, cannot be long maintained; tonus, on the other hand, is a property of sarcoplasm, and, occurring as it does without any great chemical change, can be long kept up.

Jotzko claims that his view of muscle structure throws some light on certain pathological conditions, as, for instance, the atony of muscles in neurasthenia; this is probably due to the loss of some sarcoplasmic excitant (suprarenal internal secretion); as also in hysterical contractions the absence of fatigue of the part and

the fact that the temperature of the contracting muscles is not raised seems to point to some undue excitement of the sarcoplasmic elements of the muscles.

HEWAT FRASER

# THE MYOGRAPHIC AND THE ERGOGRAPHIC CURVE

(259) (1) Z. TREVES and (2) E. BELMONDO, *Riv. di Patolog. nerv. e ment.*, Vol. ix. f. 5, May 1904, p. 237.

THESE two papers continue the controversy which has arisen between Dr Treves and Professor Belmondo with reference to Dr Lugiato's studies on the form of the ergographic curve.

Dr Treves considers that the parallel which Dr Lugiato has drawn between the myographic and the ergographic curves cannot be sustained, and that his transference of the nomenclature of the former to the latter is illegitimate and productive of confusion.

The ergograph is essentially a machine which transforms, with variable result, the energy emitted by the muscle into external work. It writes never the "muscular curve," but always the curve of the movement assumed by the instrument. Even if it were brought to perfection so as to interfere in no way with the normal functioning of the bony levers, still these levers themselves would substantially modify the external effect in comparison with that which would be produced were the muscle free to execute its own contraction, as in the myographic curve.

Again, the mechanical results produced in the ergograph are due to the algebraic summation of rhythmic contractions of opposing muscles, and, moreover, in the case of voluntary work we are ignorant of the influence which the appreciation of the external circumstances of work exercises upon the automatic part of the functioning of our muscles. For these and other reasons it is a mistake to attempt to use the ergograph to register the "muscular curve."

Professor Belmondo replies by a brief defence of Dr Lugiato's methods and conclusions, and adds that it appears to him the controversy should terminate by the recognition on the part of Dr Treves of these two axioms: (1) That the laws of muscular contraction studied by the usual myographic methods in various animals are far from expressing the true process by which the muscles contract in their natural environment. (2) That the difficulty of research in the case of human muscles in the given conditions is greater in every case than Dr Treves appears to believe, and that therefore we must so far content ourselves with apparatus which, as he has always admitted for the ergograph, gives results which are not rigorously exact.



The first of these propositions Professor Belmondo illustrated in a previous contribution. He devotes the rest of the present paper to a brief development of the second.

MARGARET DRUMMOND.

# **INFLUENCE OF ELECTRICITY ON THE CEREBRAL PULSE.**

(260) E. GENTILE, *Ann. d'Électrobiol. et de Radiol.*, Vol. vi. No. 5, p. 553.

LOWENFELD in rabbits, Sgobbo, Rumpf, Fisher, Sollier and Capriati in the case of the human subject, have, with varying results, studied the effect of electricity on the cerebral pulse.

Gentile, after careful observations on two men with exposed cerebral surface, concludes that the effects of the application of faradic and galvanic currents vary greatly at different times, both as regards the pulse and the regulation of the vascular supply of the brain.

A. DINGWALL FORDYCE.

## **PATHOLOGY,**

# **ON THE NATURE AND PATHOGENY OF RADICULAR LESIONS (261) OF THE CORD WHICH ACCOMPANY CEREBRAL TUMOURS.**

J. NAGEOTTE, *Rev. Neurolog.*, Jan. 1904, p. 1.

THE author has had the opportunity of studying the spinal cord changes in three cases of cerebral tumour, and he has been able to satisfy himself that a process analogous to that found in the course of chronic syphilitic meningitis occurs in the posterior roots in cases of cerebral tumour. An inflammatory focus develops on that portion of the course of the root which has received the name of "nerf radiculaire," and is figured in the diagram given as that portion of the posterior root which lies immediately on the proximal side of the ganglion. The author has further been able to prove a similar lesion on the anterior roots, which are sometimes affected like the posterior roots.

Three cases are then described, in the first of which degeneration of the posterior columns was due to a specific lesion, while in the other two cases large intracranial tumours were present. The situation of the lesion to which the degeneration of the posterior roots is attributed is represented at a point on the proximal side of the posterior root ganglion, and is figured in the paper as an actual swelling visible to the naked eye. The lesion of the posterior columns which occurs in cerebral tumours resemble that found in cases of tabes—

1. By their markedly radicular character.
  2. By their relation with an inflammatory focus situated on the roots.
  3. By their starting in the distal part of the neuron affected.
- The lesion differs from that found in tabes—
1. By the non-systematic distribution of the degeneration although radicular in origin.
  2. By the non-syphilitic nature of the inflammatory focus on the roots; and
  3. By a less marked tendency of the process to destruction of the nerve fibres.
- F. E. BATTEN.

**CONGENITAL CYSTS OF THE FOURTH VENTRICLE.** By (262) J. RAMSAY HUNT, M.D., *Amer. Journ. Med. Sciences*, March 1904.

Cysts in the region of the fourth ventricle are classified as follows:—

1. Parasitic cysts.
2. Cystic degeneration of a new growth.
3. Cystic degeneration of the choroid plexus.
4. Cystic dilatation of the fourth ventricle from occlusion of its communications.
5. Congenital cysts.

Two cases are reported of brain tumour in young people associated with congenital cysts of the brain stem projecting into the fourth ventricle. The first case was that of a boy aged seven years, who developed symptoms referable to a tumour of the optic thalamus. At the autopsy a large mixed-celled sarcoma was found situated in the left thalamus and extending to the basilar surface of the pons. The fourth ventricle was distended by a large cyst, which was firmly attached to its floor, and, penetrating the substance of the pons, terminated in the midst of the neoplasm.

The second case was that of a boy aged seventeen years, who developed symptoms referable to a tumour in the right crus cerebri. The autopsy revealed a glioma of the right crus cerebri, which extended into the pons. A cyst of the fourth ventricle penetrating its floor, perforating the substance of the pons, and distinct from the aqueduct, and appearing on the under surface of the right crus, was in connection with the tumour mass.

The writer considers that these cysts were of congenital origin because:—

1. The cyst wall throughout was composed of medullateral nerve fibres and glia.
2. The cysts were not intercalated, but formed an integral part

of the pons, and the relations of the cyst wall to the adjacent nervous tissue were intimate.

3. The cysts showed remnants of an endothelial lining.
4. There were no cerebral symptoms preceding the development of the tumours.

JAMES COLLIER.

**THE EXAMINATION OF THE TISSUES OF THE CASE OF SLEEP-  
(263) ING SICKNESS IN A EUROPEAN.** G. C. Low and F.  
WALKER MOTT, *Brit. Med. Journ.*, April 30, 1904, p. 124.

In the *British Medical Journal* of April 30th, 1904, Mott and Low describe the examination of the tissues of a case of sleeping sickness in a European. The wife of a Congo missionary consulted Sir Patrick Manson in 1902 for chronic pyrexia and enlargement of the spleen. Trypanosomes were afterwards found in the blood, while the patient was in the wards of the London School of Tropical Medicine. After going home to Bristol some signs of drowsiness appeared, and symptoms of sleeping sickness developed.

She died in a state of coma. The usual microscopic changes of sleeping sickness were found in the brain and other organs, infiltration of the pia arachnoid with large and small called mononuclear leucocytes, especially marked over the cerebellum and medulla, infiltration of mononuclear leucocytes round the small vessels in the cortex and other parts of the brain, similar changes in the spinal cord and degeneration of nerve cells. The ganglion cells showed acute changes probably of the nature of a coagulation necrosis. There was chromatolysis, the Nissel granules being either indistinct, absent, or a dust-like powder. The neuroglia cells in places showed active proliferation. In addition to those changes the epicardium showed a marked mononuclear infiltration round the vessels, with early pericarditis. There was a general invasion of diplococci; only one evidence, however, of a trypanosome was found in the large number of sections examined.

AUTHOR'S ABSTRACT.

**CLINICAL NEUROLOGY.**

**TRAUMA IN RELATION TO DISEASE OF THE NERVOUS  
(264) SYSTEM.** JUDSON S. BURY, *Brit. Med. Journ.*, April 30, 1904,  
p. 997.

THE writer considers that though neurasthenia and hysteria following severe accidents have received sufficient attention, there ought to be a fuller recognition of the prolonged and sometimes permanent incapacity for work that may result from severe shaking as produced by blows, and falls upon the head and body. It is

in this limited sense that his present paper deals with trauma. He considers the subject under the heads of injury to the brain, to the cord, and to the peripheral nerves.

*Concussion of the brain.*—The pathological results are various, including hæmorrhage from the membranes or brain, inflammatory changes of various types, laceration of the brain, and new growths. The symptoms are referable to the following results.

*Meningeal hæmorrhage.*—Two cases are recorded. In one of these the man, injured by a fall, remained quite well a week after the accident, becoming then gradually comatose and dying at the end of 3½ weeks, when a large clot was found covering the whole left Rolandic area. In the other case a man who had fallen upon the back of his head went about as usual for 16 months, suffering from headache and loss of memory, the cause proving at the end of that time to be a large membranous clot beneath the dura. In these and similar cases the writer prefers to attribute the serious and delayed symptoms to degeneration following bruising of the brain, not to the subdural clot, which, when it produces symptoms, causes stupor or coma.

*Neurasthenia* he treats at considerable length, dividing the cases into two classes, one due to immediate bruising of the brain in which mental dulness, apathy and drowsiness are present from the first; and a secondary group resembling non-traumatic neurasthenia in its symptoms, and coming on in those of neurotic heredity as the result of ready molecular disintegration, cell degeneration, and consequent surrounding sclerosis. He notes the intolerance of all these cases for alcoholic drinks, and he wishes to lay particular stress on these "minor degrees of mental disturbance which are apt to be ignored, and yet seriously interfere with the patient's wage-earning capacity."

*Insanity* occasionally arises and may present a close resemblance to general paralysis.

*Cerebral tumour* is an occasional result, and one case has been recorded by the writer.

*Late apoplexy*, some weeks after a blow on the head, has been much studied in Germany, and is attributed to necrotic softening with degeneration of arterial walls.

*Concussion of the spinal cord* from a blow on the back, head, feet, or buttocks, often results in suddenly developed spastic paraplegia due to myelitis or softening without any injury of the bones, and the writer refers to several cases. From this and from references to numerous "intermediate" cases in which grave symptoms came on days or weeks after the injury, he induces the theory that many cases of chronic nervous disease are due to long forgotten trauma. He records two cases of disseminated sclerosis in which this connection seems sufficiently obvious.

*Injury to the periphery of the body* he considers may in some cases result in disease of the spinal cord. He bases this suggestion upon the well-known atrophy of muscles which occurs in the proximity of an injured joint, and which was shown by Raymond not to occur when the posterior nerve roots were divided. As these cases of arthritic muscular atrophy are therefore due to afferent influences, he suggests that many cases of progressive muscular atrophy appear to result from a sprain or other injury to a limb. As a practical conclusion, the writer believes that a longer rest in bed after injuries than is usual in hospital practice would do much to reduce the number of sufferers from chronic diseases of the nervous system.

JOHN D. COMRIE.

**INTRADURAL TUMOUR OF THE CERVICAL MENINGES, WITH  
(265) EARLY RESTORATION OF FUNCTION IN THE CORD  
AFTER REMOVAL OF THE TUMOUR. HARVEY CUSHING,  
*Annals of Surg.*, June 1904.**

THE patient, a man aged 30, was admitted to the Johns Hopkins Hospital under the care of Dr Osler on October 14, 1903, complaining of "pain in his shoulder and of awkwardness in his gait." The family history was unimportant. The patient had "always been healthy." He denied venereal disease and there was no history suggestive of syphilis. In July 1902, the patient began to have pain in the flexor surface of his left forearm, later in the left shoulder and upper part of back. The pain was often very severe and was aggravated by movements of the neck. Sneezing, laughing, yawning, or coughing caused the pain to shoot into the arm. Recently the pain had been less severe. Early in 1903 a burning sensation was experienced in the right and some weakness in the left leg. For some months he had noticed that the muscles of the left hand had been wasting and that differences in temperature were less easily recognised in the right leg. The bowels had been constipated and there had been some hesitancy in micturition.

The following are some of the more striking positive facts ascertained on examination:—The patient carried his head somewhat stiffly. Passive flexion of the neck is strongly resisted. There was no thickening, prominence, or tenderness of the cervical spines. There was marked atrophy of the intrinsic muscles of the left hand, with weakness of the extensors and flexors of the fingers and of the flexors of the wrist; less so in the wrist extensors. The supinators and pronators, flexors and extensors of the forearm, pectoral and latissimus dorsi are also weak, as is the entire musculature of the left side of the body below this level and the left lower

limb; although the patient could stand and walk, he quickly tired. On admission, thermic and pain perception was lost over the right side of the body below the second intercostal space, but there was no impairment of tactile sensation. At a later date the thermo-anæsthesia and analgesia extended along the inner side of the right arm and forearm, involving the last three fingers. Posteriorly it reached as high as the first thoracic vertebra. There was in addition some relative thermo-anæsthesia and analgesia on the right side from the third to the fourth ribs. The knee-jerks were exaggerated on both sides and there was double ankle-clonus especially marked on the left. The plantar reflexes were very active, but there was not a characteristic Babinski sign. The tuberculin test gave a negative result.

A laminectomy was performed under ether on November 19th. The laminae of the two lower cervical and first dorsal vertebrae were removed. The dura was found to be abnormally tense and vascular, the median posterior vessels being unusually dilated. When the dura was opened the arachnoid bulged into the opening like a distended bubble, fluid spurting out in jerks when it was pricked. An oval tumour of a dusky purplish colour was seen when the arachnoid collapsed, pressing on the left side of the cord, and producing considerable flattening. A posterior root lying over the tumour was divided. An area of partial analgesia and thermo-anæsthesia was present over the posterior and outer side of the upper arm and down the back of the forearm to the wrist for some days after the operation. It was necessary to remove the laminae of the fifth cervical vertebra to obtain complete access to the tumour, which was somewhat adherent laterally to the membranes. The removal of the tumour caused some bleeding, which was readily controlled by pledgets of sterile absorbent wool. The wound was closed in layers, no drainage being used; a dressing with plaster of Paris support for the head and neck was applied. The operation was unattended with shock.

On the day following the operation the motor condition had much improved, the anæsthesia had considerably diminished in degree, and the ankle-clonus on the right side had disappeared. The patient made an uneventful recovery and was discharged on December 16th, the only evidence of pre-existing trouble being the slight wasting of the left hand. Two months later he "felt as strong as ever before," there was no difference in the appearance of the hands, and the only physical sign which remained was some exaggeration of the deep reflexes. On microscopical examination the growth was found to be a fibrosarcoma.

A summary of the reported cases of intradural tumour which have been successfully operated on is appended. The paper is well illustrated.

EDWIN BRAMWELL.

**MULTIPLE SCLEROSIS: ITS OCCURRENCE AND ETIOLOGY.**

(266) By SMITH ELY JELLIFFE, M.D., Ph.D., *Journal of Nervous and Mental Diseases*, July 1904, p. 446.

IN this paper Dr Smith Ely Jelliffe analyses all the cases of multiple sclerosis, which have been definitely diagnosed as such, in Dr M. Allen Starr's neurological clinic during the past sixteen years. During this period there were 31,502 patients suffering from some form of nervous or mental trouble; of these, 109 patients presented the symptoms held to be diagnostic of multiple sclerosis. In no case was a diagnosis of multiple sclerosis made without the presence of three of the four cardinal principles—increased reflexes, intention tremor, nystagmus and scanning speech. Consequently 1 in every 300 cases of nervous disease, functional and organic, was a case of multiple sclerosis. Dr Jelliffe contrasts these figures with those of other observers, and states that: "Certainly our own figures would justify Dr Bramwell's assertion that multiple sclerosis is more common in England than in the United States, and it seems reasonably certain that we make a diagnosis of the disease on much the same lines as our English confrères." Of the 109 patients, 47 only were of American birth, and even amongst these at least 12 were of the second generation of foreign stock. The nationality of the patients was as follows: United States, 47; Germany, 20; British Isles, 19—17 being Irish; Austria, 6; Sweden and Norway, 5; Russia, 4; Cuba, Italy and coloured, each 1; unknown or untraceable, 4.

Of the 109 cases, 68 were males and 41 females. Dr Jelliffe states that most modern writers are unable to detect any difference in the incidence of the disease in the two sexes, though Charcot maintained that multiple sclerosis was more frequent in women than in men. (In 110 cases which have been analysed and studied by the abstractor, 67 were females and 43 were males.)

Of the 109 cases, 31 men and 15 women, or 46 in all (less than one-half), occurred between the ages of 18 and 35; 17 (8 males and 9 females) were younger; 43 (27 males and 16 females) were over 35 years of age.

Occupation seemed to have no influence on the production of the disease. Only 8 of the 109 patients showed distinct nervous inheritance. As regards direct causation, Dr Jelliffe thinks that three factors at least are worth inquiring into, namely, acute infectious disease, trauma and poisoning. He agrees with Hoffmann's opinion that infection undoubtedly plays an important rôle in the production of multiple sclerosis; but states that his own figures are not very convincing in regard to this point, for in

about 50 per cent. of the cases the observer made no note of having inquired into the subject. In 14 of the remaining 55 patients there was a distinct history of antecedent infectious disease. It is striking that 5 of these patients had malaria. Syphilis was found in 2, tuberculosis in 1, acute articular rheumatism 1, erysipelas 1, influenza 1, diphtheria 1, pneumonia 1, and one unknown febrile affection.

In the 109 cases, trauma was present in 13.

Although three of the 109 patients were painters, none of them had ever had symptoms of chronic lead poisoning.

Concerning other factors, his statistics showed anæmia 2, sexual excesses 1, epilepsy 1, business troubles 1, and in 2 the history of a highly strung nervous condition was recorded.

BYROM BRAMWELL.

**MULTIPLE SCLEROSIS WITH DEMENTIA.** J. R. HUNT, *Am.* (267) *Journ. Med. Sc.*, Dec. 1903.

THE writer describes the clinical and post-mortem appearances of a case in which the two conditions of multiple sclerosis and general paralysis coincided. The symptoms were at first those of multiple sclerosis; upon these supervened mental enfeeblement, progressing to dementia, and almost complete paraplegia. The duration was eight years. Post-mortem examination revealed the characteristic changes of general paralysis in addition to disseminated plaques of sclerosis in the brain, cerebellum, pons, medulla and cord.

The writer, after giving brief synopses of seven similar cases recorded by other authors, remarks upon the difficulty of diagnosing such cases, since in the early stages of each of these affections the symptoms may be very much alike. He states that in none of the cases quoted was the condition recognised clinically, and when it is remembered how complex may be the symptomatology of each of these diseases, it is not surprising that great difficulty should be experienced where they are coincident.

Finally he remarks that cases which are characterised by progressive mental enfeeblement, accompanied by mental alienation and by the somatic signs of multiple sclerosis, may be due to: (1) an extreme cerebral manifestation of the sclerotic process, of the same nature histologically as the disseminated plaques in the cord; (2) a combination of general paralysis and multiple sclerosis in the same subject; (3) a diffuse gliosis of the cortex with optic atrophy and degeneration of the posterior columns of the cord.

A. F. TREDGOLD.



**A YEAR'S LUMBAR PUNCTURES.** A. CHAUFFARD and L. BOLDIN,  
(268) *Gaz. des hop.*, June 28, 1904, p. 725.

THE authors give the results of the punctures in 140 cases in Chauffard's wards, 223 punctures being made; both from the diagnostic and prognostic point of view much information was gained from the procedure. The therapeutic result was excellent in cases of meningitis—syphilitic, tubercular and epidemic cerebro-spinal, in one case of labyrinthine dizziness, in one of renal headache with blindness and vomiting, and in one of headache after a severe herpes.

In three cases of mumps the lymphocytes in the fluid disclosed the meningeal irritation which was causing headache and bradycardia. In various febrile diseases the examination of the cerebro-spinal fluid gave definiteness to the diagnosis. Lumbar puncture also enabled the authors to diagnose four cases of purely meningeal hæmorrhage presenting the clinical symptoms respectively of diabetic coma, cerebro-spinal meningitis, uræmia; a distinction is made between cerebro-meningeal hæmorrhage due to the extension to the membranes of a deeper focus and purely meningeal hæmorrhage. Other interesting cases were three patients with Argyll-Robertson pupil and no lymphocytosis of the fluid; in two out of eleven tabetics there was no lymphocytosis.

C. MACFIE CAMPBELL.

**A CONTRIBUTION TO THE SYMPTOMATOLOGY OF ENCEPHALITIS.** W. SPIELMEYER, *Centralbl. f. Nervenh. u. Psych.*,  
(269) June 1904.

SPIELMEYER devotes this paper especially to the epileptic form of encephalitis, as contrasted with those forms where the general comatose condition or a monoplegic or hemiplegic condition is the most striking feature in the case. In those cases of encephalitis where epileptiform attacks dominate the clinical picture, the convulsions may be general or of the Jacksonian type, and suggest the diagnosis of brain-tumour; the diagnosis is the more difficult, as both in encephalitis and brain-tumour one may have the same mental symptoms; in both too there is the tendency to the development of the status epilepticus. The onset, however, is different; the encephalitis develops quickly after short prodromatal symptoms, attacks usually healthy young people, and can often be traced to some previous infectious disease; while brain-tumour has a more insidious onset and progressive course, and usually a choked disc by the time general symptoms have appeared. The author gives details of a case personally observed.

C. MACFIE CAMPBELL.

**A CASE OF CEREBRAL HEMIPLEGIA WITH PERSISTENT (270) HEMIANÆSTHESIA.** ED. LONG (de Genève), *Rev. Neurol.*, Feb. 15, 1904, p. 113.

THE author and M. Dejerine have shown from an anatomical examination of a large number of cases that it is especially in lesions of the optic thalamus that persistent hemianæsthesia is met with. The ascending sensory fibres pass to the ventral and external nuclei of the optic thalamus. From here the thalamo-cortical fibres in their further course are not located as a distinct bundle in the internal capsule, but are intimately mingled with the projection fibres which descend from the sensory motor area of the cerebral cortex. From a relatively localised lesion of the external part of the thalamus, destroying the grey substance which constitutes a relay between the ascending peduncular and thalamo-cortical fibres, a more or less complete and persistent hemianæsthesia with little or no paralysis should result.

Dejerine and Egger have recently reported two cases in which there was only a very slight hemiplegia associated with a marked anæsthesia, and in one of these cases a lesion of the optic thalamus was verified post-mortem. On account of the way in which the thalamo-cortical fibres spread after leaving the internal capsule, and the numerous collaterals which may serve as substitutes for sensory conduction, a lesion in the corona radiata and cerebral cortex has to be of much greater extent to produce hemianæsthesia. Dr Long, in his classical monograph on the central pathways for sensory conduction ("Les voies centrales de la sensibilité générale," *Thèse de Paris*, 1899, p. 146), has recorded two cases of persistent hemianæsthesia associated with hemiplegia. In the first case there was a large cortical and subcortical lesion which occupied almost the whole of the corona radiata; in the second case the lesion was cortical and very extensive. Schaffer of Budapest has recently described a case examined likewise by serial sections, in which interruption of the thalamo-cortical fibres produced a persistent hemianæsthesia. In the case described by Long in this paper there was a complete left-sided hemiplegia. Tactile and painful sensibility was almost abolished on the left side. It was not, however, possible to pass a needle through the skin as in a hysterical hemianalgesia, for the patient complained of pain although unable to localise exactly the sensation. The patient could not distinguish between degrees of temperature from  $+60^{\circ}$  to  $-5^{\circ}$ ; if, however, the glass containing ice was left for long in contact with the skin, he complained of a painful sensation which at times was recognised as cold. He was able quite easily to find either ear and the right knee with his right hand, but he could not find his left hand or knee; he said he did not know

where they were. There was left lateral hemianopia. There was defective hearing, smell and taste on both sides. The hemianæsthesia did not diminish during the eighteen months the patient was under observation in the Salpêtrière; it had been present for ten years previous to this.

These symptoms might have been accounted for by a single central lesion destroying in the lower capsular region the projection fibres from the motor cortex, the infero-external part of the thalamus, and the optic radiations where they arrived at the geniculate body and pulvinar.

At the autopsy, however, multiple lesions were found. The hemianopsia was dependent upon a small focus of softening involving the radiations of the external geniculate body, which was notably atrophied. Dr Long remarks that this case supports the theory which regards the external geniculate body as the only connection of the retinal fibres, and it shows that a very localised lesion of this nucleus or its cortical radiations may produce a definite hemianopia. The hemiplegia and hemianæsthesia were found to depend upon two areas of softening produced by vascular occlusion; one artery which was blocked was the artery of the ascending frontal convolution which supplies also the foot and anterior surface of the ascending parietal convolution. The superior part of these convolutions supplied by the anterior cerebral artery was not involved. The other was a central artery, one of the external striate arteries which supplies the external aspect of the lenticular nucleus and the external capsule. This case demonstrates the important fact that destruction of the thalamo-cortical segment of the sensory paths beyond the capsule is able to produce a hemianæsthesia marked in degree and persistent.

EDWIN BRAMWELL.

**A CASE OF SOLITARY TUBEROLE OF THE PONS: REMARKS  
(271) ON THE PATHWAY FOR SENSATIONS OF TASTE  
FROM THE ANTERIOR PORTION OF THE TONGUE, ETC**  
CHARLES S. POTTS and WILLIAM G. SPILLER, *Univ. of Pennsylvania Medical Bulletin*, December 1903.

THE case was that of a man, aged 55 years, with marked weakness of the right arm and leg; weakness of the muscles of the left side of the face, excepting the orbicularis palpebrarum; of the muscles of mastication on the left side; fibrillary tremors of these muscles; weakness of the left external rectus, with the loss of the power of associated movement of the eyes to the left, but with preservation of the power of convergence. Diminution of the power of appreciating touch, pain, heat and cold

on the right arm, leg, side of the trunk, neck, occipital region and ear. On this side also were astereognosis and loss of the sense of position. Diminution of the power of recognising touch, pain, and heat and cold on the left side of the face and head. Slight loss of power of recognising heat and cold on the right side of the face. Anæsthesia of the conjunctiva, mucous membrane of the nose, mouth and tongue on the left side; also deafness in left ear and loss of sense of taste in the left anterior half of the tongue. Some ataxia of both legs, most marked in the right. A tuberculoma of the left side of the pons found at the autopsy.

Dr Potts, in his remarks upon the pathway for the taste fibres, states that the fact that the sense of taste was affected only on the anterior portion of the left side of the tongue, would seem to bear witness to the view that taste sensations from this portion of the tongue reach the brain by way of the fifth nerve, and not by the glosso-pharyngeal. He is of opinion that the fact that the posterior longitudinal fasciculus was involved in the case tends to disprove the view that the nuclear origin of the fibres which supply the orbicularis palpebrarum is in the nucleus of the third nerve, and that by way of this tract they join the fibres of the seventh nerve after the latter have left the facial nucleus.

Dr Spiller discusses the paralysis of lateral conjugate movement of the eyes which was present in the case. He believes that this is a very important symptom in differentiating extra and intra-pontine tumours, although the absence of paralysis of lateral conjugate movement is no proof that the pons is not invaded, but merely that the posterior longitudinal fasciculus is intact.

EDWIN BRAMWELL.

#### **INTRACRANIAL PSAMMO-SARCOMA WITHOUT PARALYSIS.**

(272) WALTER D. BERRY, M.D., *Amer. Journal of Insanity*, Vol. lx. No. 4.

THE case reported is that of a woman who, at the age of 50 years, suffered a severe blow on the frontal region. No immediate sequelæ were noticed, but five years later she suffered with headaches and her memory began to fail. At the age of 66 years an attack of influenza was followed by temporary mental derangement, and three years later another attack of influenza was followed by permanent insanity and she was admitted into the State Hospital at Waterbury with chronic delusional insanity. There were never at any time symptoms indicative of local cerebral disease. She died of pneumonia at the age of 74 years.

At the autopsy a hard tumour the size of a large apple was found growing from the dura mater at a point 4 cm. to the right

of the junction of the sagittal and coronal sutures. It pressed deeply into the brain, and was limited behind and externally by the second frontal gyrus and mesially by the middle line, the paracentral lobule being much compressed. The right caudate nucleus and right internal capsule were much compressed. The tumour proved to be a typical psammoma.

The writer draws attention to the rarity of the presence of intracranial tumours amongst asylum patients.

He discusses various opinions as to the nature of the psammoma. Senn considered that it was a dural endothelioma and should be classified with the sarcomata. Bland Sutton refers to it as a choroid plexus epithelioma.

Ziegler considers the psammoma as a sarcoma with local calcification, while Hamilton states emphatically that it has nothing in common with the sarcomata.

Stengel thinks that the psammoma represents no distinct species of tumour growth, but rather a peculiarity of several kinds of growths. Delafield and Prudden consider it simply as a calcifying fibro-sarcoma.

The author considers the absence of paralysis in this case peculiar. He states that progressive growth in a psammoma is usually arrested by fatty and calcareous degeneration, and that if these events do not occur, growth is continuous. He draws attention to the fact that the concentric bodies consist of endothelium-like cell nests arranged "around" blood-vessels. The paper is accompanied by excellent illustrations. (The absence of paralysis in cases of slowly growing tumours of the brain of a locality such as the one instanced is no rare event. Each concentric body in a psammoma is derived from a blood-vessel by endothelial proliferation within and not round the vessel.—Rev.)

JAMES COLLIER.

**TUMOURS OF THE PONTO-MEDULLO-CEREBELLAR SPACE.**  
(273) **ACOUSTIC NEUROMATA (CENTRAL NEURO-FIBROMATOSIS).** JOSEPH FRAENKEL and J. RAMSAY HUNT, *Medical Record*, Dec. 26, 1903.

THE authors consider that the tumours not unfrequently met with upon the intracranial course of the cranial nerves are pathological occurrences identical with those of general neuro-fibromatosis of the cerebro-spinal and sympathetic nerves. They state, further, that these two conditions not unfrequently co-exist. They report three cases of unilateral tumours of the eighth nerve, one case of bilateral tumour of the eighth nerve, and one case of tumour of the fifth nerve.

Such tumours most often occur upon the eighth nerve, and not unfrequently both eighth nerves are affected. They occur less frequently upon the fifth nerve, and they are only rarely found affecting the intracranial portions of the other cranial nerves. Sometimes the tumours are multiple, and they may then occur upon different nerves.

Hyperplasia of the cortical glia and dural endotheliomata and psammomata are not unfrequently associated with these tumours, and in more than one reported case intracerebral sarcomata have co-existed.

Histologically, these tumours are nearly always typical neuro-fibromata, but occasionally they are heterogeneous and show sarcomatous and gliomatous elements.

The intracranial neuro-fibromata vary in size from that of a cherry to that of a hen's egg. They are hard, tough, and always encapsulated. The surface of the growth is nodular and irregular.

The point of origin of the growths is undecided, and the authors are content to mention Sternberg's belief that they arise from embryonal rests in the so-called dorsal cranial nerves.

The symptomatology is that of tumours of the posterior fossa, the valuable distinctive sign being the initial implication of one cranial nerve.

The removal of these tumours is difficult on account of their situation, yet their very loose attachment to surrounding structures favours surgical interference, which affords the only hope in prognosis.

The cases are reported in great detail and are admirably illustrated. Of especial interest is the association of dural endotheliomata and psammomata in one of them.

JAMES COLLIER.

**ON THE RELATIONS OF EPILEPSY TO AMENTIA.** TREDGOLD, (274) *Brit. Journ. Children's Dis.*, July 1904, p. 291.

In this paper the author discusses the connection existing between epilepsy and mental deficiency, basing his remarks upon the examination of nearly 600 cases of amentia, in which he specially enquired into the presence or absence of convulsions. Every variety and degree of amentia was included, from the simply feeble-minded to the gross idiot.

He remarks that it has been usual to class all such patients together as "epileptic" idiots or imbeciles, whereas examination shows them to belong to several entirely distinct groups. The relationship existing between epilepsy and amentia is in fact three-fold, as follows:—

- (1) Primary amentia in which epilepsy occurs as a mere complication.
- (2) Idiopathic epilepsy causing amentia.
- (3) Gross cerebral disease causing epilepsy and amentia.

In *group 1* the epilepsy has no causal relationship to the mental defect; but is simply a complication to which the imperfectly developed brain would appear to be more than usually prone, since no less than 36 per cent. of all primary aments are epileptic. The prevalence of epilepsy, moreover, varies with the degree of mental deficiency, and convulsions are present in 11 per cent. of feeble-minded, 42 per cent. of imbeciles, and 56 per cent. of idiots. Of those cases where a gross cerebral lesion co-exists, 70 per cent. are epileptic.

In *group 2* the epilepsy is the actual cause of the arrested mental development. As is well known, frequently repeated and severe epileptic convulsions often result in dementia; but if, in addition, the onset of the fits takes place in early life, before neuron development is complete, they may also cause amentia; the anatomical substratum of amentia being an imperfect or arrested development of the same cortical neurones as undergo degeneration in dementia. In the cases of this group, therefore, the mental defect is in reality an incidental phase of a degenerative process, and the chances of improvement under educational methods are extremely slight.

In *group 3* the essential condition is a coarse cerebral lesion of which both the epilepsy and amentia are symptomatic, and prior to the occurrence of which the mental development of the patient has been normal. The pathological processes which give rise to these lesions are many and varied, but are divisible into two classes, viz., *vascular* (asphyxia neonatorum, trauma, whooping-cough, etc.) and *toxic* (scarlet fever, measles, small-pox, typhoid, otitis, rhinitis, polio-encephalitis, etc.). The final forms assumed by the lesions being localised atrophy, softening or sclerosis, cysts, chronic meningo-encephalitis, porencephalus, and hydrocephalus.

The author remarks that many children in whom these cerebral affections occur die, others appear to recover completely, whilst in yet others there results amentia, epilepsy and paralysis, either singly or in various combinations. It is impossible to predict which of these will occur in any particular case, but such factors as (1) the time of occurrence, (2) the site, (3) the extent, and (4) the nature of the lesion, will determine the result. With regard to the time of occurrence, he states that the brain normally contains a number of neuroblasts which under ordinary circumstances never attain maturity, that, in fact, there is a potentiality of cerebral development to which the ordinary individual does not attain, and he adduces reasons for the view that in the event

of a cerebral lesion occurring in early life, these neuroblasts may, under certain circumstances, be called into play, and so bring about compensation. This compensation, however, will also be dependent upon the site, extent and nature of the pathological process: where the latter remains active, compensation is unlikely to occur; but where it becomes stationary, special educational methods may to a greater or lesser extent repair the disease.

The writer states that in the majority of these cases of cerebral lesion, even where they are unilateral, the fits are typically epileptic. In a few, however, they are Jacksonian; but since these in course of time tend to become generalised and to be attended with loss of consciousness, it is probable that if the patients could be seen sufficiently early a greater proportion would be found to be epileptiform at the beginning. He alludes to the different prognoses of the three groups, and concludes with a table showing the chief points of difference between them.

AUTHOR'S ABSTRACT.

**A CASE OF APHASIA WITH LOSS OF MEMORY OF NOUNS (275) (SENSORY ANOMIA) WITH AUTOPSY.** CHARLES L. DANA and JOSEPH FRAENKEL, *Journ. Nerv. and Ment. Dis.*, Jan. 1904, p. 15.

THE case here described is that of a man who, in addition to other symptoms was unable to name things which he saw, touched, tasted or smelled, although he was able to describe their uses in each instance. He could understand spoken words and could read aloud and understand. He could repeat the names of things, and he could recall objects whose names he has seen. He could talk voluntarily and had a good vocabulary, using a good many nouns, although he was sometimes at a loss for them. His speech was sometimes paraphasic, but there was no jargon aphasia. With regard to his writing capacity it was difficult to speak positively, for he could never write well. He could copy.

At the autopsy, which was made by Dr J. R. Hunt, three distinct patches of superficial softening were to be seen upon the internal surface of the left cerebral hemisphere. One of these, which involved the posterior third of the first temporal convolution, extended into the lip of the Sylvian fissure and measured two cm. antero-posteriorly by two cm. vertically; another area of softening lay a little anteriorly to the middle third of the second frontal convolution; the third was situated on the inferior surface of the first frontal. Upon sectioning the brain a small spot of subcortical softening about one cm. in diameter was found on the third temporal convolution just below the level of the cortical softening mentioned above.



The situation of these areas of softening is illustrated by photographs.

The authors discuss anomic aphasia and the question of a naming centre, and come to the following conclusions:—

1. There is no special centre for naming. There is an important station in the sensory mechanism for naming concrete things, and it lies in the first and second temporal convolutions, and this is a centre for sensory anomia.

2. Sensory anomia is a form of aphasia characterised by inability to name things seen, and often things heard, felt, tasted or smelled. Its especial character can be indicated in particular cases by the terms optic, optic and tactile, etc.

3. The use of anatomical terms, such as cortical and sub-cortical, to indicate psychological processes, is not a very practicable or rational procedure; and it would be better to drop the words cortical and subcortical in describing aphasias. In the present case there was what would be called a subcortical aphasia with a lesion mainly cortical. EDWIN BRAMWELL.

**ON GANSER'S SYMPTOM.** By HENNEBERG (of Berlin), *Centralbl. f. (276) Nervenh. u. Psych.*, 15th June 1904, p. 388.

HENNEBERG bases his views on the results of twenty-five observations. He distinguishes Ganser's symptom in the narrow sense from Ganser's symptom complex or confusional state. The symptom consists in the patient giving false answers to simple questions, but always with a certain relevance in the answer. The catatonic, on the other hand, gives answers with little or no relation to the question. The symptom is seen in hysterical and hypochondriacal patients and may appear after an ordinary hysterical attack. The majority of cases observed were cases of hysterical psychosis in criminals. The course of the cases put catatonia out of the question. No particular diagnostic importance is to be attached to the symptom, which is not confined to hysterical psychoses. C. MACFIE CAMPBELL.

**THE PATHOGENESIS OF NEURASTHENIC HEADACHE.** L. (277) MIÉCAMP, *Arch. gén. de méd.*, June 28, 1904.

WITH regard to the anatomical seat of the process causing the headache, Miécamp considers that it is the cortical nerve cell in general, and the sympathetic system which are affected. The headache is the manifestation of a disordered condition of the nerve cell, brought about by auto-intoxication or by a primary disorder of the sympathetic; the symptom is associated with vaso-motor disturbances in proportion to its severity. C. MACFIE CAMPBELL.

**THE VALUE OF THE RÖNTGEN RAYS IN NEUROLOGY.**  
(278) V. RUTKOWSKI, *Charité-Annalen*, 1904.

DR RUTKOWSKI describes twelve cases, and gives photographs taken from them.

The first case is one of acromegaly. Then follow five cases of spinal caries, three in the dorsal and two in the lumbar region, photos of which are taken in the direct antero-posterior diameter. Several of these photographs are good, while some are indifferent. In every case an accurate diagnosis was made without the help of the rays, which, however, have served to provide interest in this prettily illustrated paper.

In two cases of tumour of the brain, photographs are given revealing nothing, but in a case of poliomyelitis anterior acuta the bone atrophy is clearly shown; while in two cases of supernumerary toe, we see naturally the extra bones. Two good photographs—lateral and antero-posterior—are presented, showing the position of bullets in the brain.

A. DINGWALL FORDYCE

**PSYCHIATRY.**

**THE METAPHYSICAL CONCEPTION OF INSANITY.** J. H.  
(279) LLOYD, *Journ. Nerv. and Ment. Dis.*, June 1904.

LLOYD calls attention to the abstract and to metaphysical conceptions of insanity embodied in legal opinions. Legal tests for insanity have often been based on the old division of the mind into faculties; these were looked on as separate entities, each liable to be attacked separately, and thus give a definite form of insanity such as moral insanity. Insanity, on the contrary, is a disease of the organic brain cells, and not of abstract mental qualities.

C. MACFIE CAMPBELL

**UNILATERAL HALLUCINATIONS OF HEARING.** E. LUGARO,  
(280) *Riv. di Patol. nerv. e ment.*, Vol. ix. f. 5, May 1904, p. 228.

THE author states that it is an established fact that lesions of the peripheral auditory apparatus have some importance in the pathogenesis of auditory hallucinations, and refers to various cases which support this statement. Of these the most demonstrative are those where a unilateral lesion coincides with unilateral symptoms upon the corresponding side.

A case of this kind is described in considerable detail. The patient was a man of 39. There was little of importance in his family history, but an aunt suffered for ten years from delusions of persecution, and one of his three sisters was the subject of hallucinations of hearing, which appeared after the death of a

relative. The patient himself had scarlet fever at 7, and contracted syphilis at 25, but appeared to have been cured.

In February 1898 the patient became melancholic after hearing of his father's death, and in May attempted to commit suicide with a revolver. The projectile penetrated the right external auditory meatus, traversed the tympanic cavity, and lodged in the temporal bone, from which an attempt, partly successful, was made to remove it. In June the man was sent to an asylum, but he improved so rapidly that, after twenty-four days, he was liberated, in charge of his wife. When asked why he had attempted suicide, he replied that he could not think why he had been so foolish. He remembered all the events of his stay in the asylum.

In November 1901 his wife aborted, and he once more became much depressed and had to be sent to the asylum. Here he was taciturn and immobile and did not eat spontaneously, but swallowed what was put in his mouth. In a few days he became quite lucid, and then said that during the preceding days he had often heard a voice in his right ear saying, "Kill yourself, kill yourself." This voice, he thought, was that of his dead father. After leaving the asylum he continued to hear a rumbling in his ear, and after returning to work he began occasionally to hear the voice again. The voice was heard chiefly at night, or when he was alone, never when he was actually working.

By and by a new symptom appeared. The voice would repeat in his ear what he had been thinking.

In November 1903 the hallucinations became more frequent. In June an acquaintance got married, and he gave a present identical with one which had been given by the bridegroom. This made a great impression upon him, and he feared he had given offence. He went off his sleep, became depressed, and once more had to be confined in the asylum. Once more his mental condition improved rapidly and he was set at liberty; but the hallucinations of the right ear continued. While he was in the asylum he was of a sad demeanour, almost melancholic. He did not converse with the other patients, but answered readily, although slowly, when questioned. He appeared interested in his own examination.

As to the condition of his ears the right tympanic membrane was cicatrised and immobile on politizerisation; the left was opaque, but not retracted. The whispered voice was not audible on the right. The watch could be heard on contact, but only slightly. A tuning fork placed on the vertex was referred to the left.

There was slight horizontal nystagmus on extreme abduction of the eyes.

After dismissal he returned to his ordinary work. He works fairly hard, but shows little initiative. He is apathetic and in-

different, speaks little and rarely laughs. He leads a monotonous and retired life.

After discussing the case at some length, the author reaches the following conclusions. The facts of this case of unilateral auditory hallucinations associated with a lesion of the corresponding ear, but coinciding also with psychic disturbances of an eminently representative context, confirm once more the belief that hallucinations are conditioned by an abnormal activity of the supersensory, that is to say of the representative psychic centres. The peripheral lesion is a factor favourable to the production of the hallucinations, but by itself insufficient. The unilateral orientation of the hallucinations is determined by a double mechanism. In the first place the lesion tends to direct the attention to that side; and in the second, the deafness renders more sensitive the centres or groups of nerve cells corresponding to the deaf ear.

W. B. DRUMMOND.

**KORSAKOFF'S SYMPTOM COMPLEX IN ITS RELATIONS TO (281) VARIOUS DISEASES.** By BONHOEFFER (of Heidelberg), *Centrbl. f. Nervenh. u. Psych.*, 15th June 1904, p. 393.

KORSAKOFF's conception of a definite disease of alcoholic origin, neuritic symptoms, and a characteristic clinical picture, has been abandoned. The symptom complex may be of various origin and is characterised by defective power of attention, retrospective amnesia, loss of orientation in time and space, pseudo-reminiscences, ideas of greatness. These symptoms are seen to a certain extent in uncomplicated delirium tremens; but delirium may precede the syndrome although not of alcoholic origin. Hæmorrhagic encephalitis may give neuritis and amnesia. Lead or arsenic may be the etiological factor; the course of the illness is milder if due to arsenic, the saturnine clinical picture is rather different. Senile and arterio-sclerotic processes are often the cause of the syndrome, in which cases conditions of terror, necessity for continuous activity, and fainting fits are common. Tumour and concussion sometimes show the same syndrome. Usually the condition develops out of an acute mental attack, and in senile cases leaves the mental state much impaired: in other cases the recovery may be practically complete.

C. MACFIE CAMPBELL.

**CASE OF DEMENTIA PRÆCOX WITH AUTOPSY.** W. R. (282) DUNTON, *Am. Journ. Insan.*, April 1904.

DR DUNTON reports a case which he diagnosed as one of dementia præcox. The patient was a woman, æt. 37, who at the beginning

exhibited depression, delusions of persecution, self-accusation, probably visual and aural hallucinations, mental confusion and feeble attention. Physically she suffered from headache, insomnia, and loss of appetite. Later the confusion and depression decreased, frequent outbursts of excitement due to the insistence of the hallucination supervened, impulsiveness became marked, she became dirty in her habits, and dementia became gradually more marked. No systematisation of delusions could be made out, and she was evidently considerably disoriented. "The usual accompanying symptoms of this later stage of dementia præcox, such as stereotypy, negativism and irrelevant answers were present, and impulsiveness became more marked." Finally, the patient became more resistive, more demented, and died. The microscopical examination of nerve cells of convolutions showed: increase of yellow pigment, frequent central chromolysis, frequent swelling of cells, nuclei atrophied, displaced and distorted, occasional neurophagocytosis, and increase of glial nuclei about cells. In the cerebellum, decrease in number of Purkinje cells, and those remaining were "ghost cells," atrophy and distortion of nuclei and disintegration of chromoplasm. The writer also mentions that the finding of similar changes in animals dying of experimental toxæmia is confirmatory of theories advanced by Nolan, Bernstein, and others, that the condition is caused by an auto-intoxication, probably emanating from the reproductive organs, and concludes that in dementia præcox we are dealing with a degenerative psychosis, probably of autotoxic origin and of slow course. With regard to the diagnosis of this case, the reporter should mention that the stereotypy and negativism mentioned above as being present receive but the most slender support in the clinical notes in the text.

R. CUNYNGHAM BROWN.

**HYSTERICAL INSANITY.** By RÆCKE, *Centralbl. f. Nervenk. u. Psych.*, 15th June 1904, p. 394.

RÆCKE puts the percentage of hysterical insanity at from 4 to 6 per cent., as against the 1 to 3 per cent. of Hess. He agrees with Binswanger that a psychosis can only definitely be called hysterical if it develops directly out of simpler elements of certain hysterical nature. He proceeds to define these elementary psychic symptoms of hysterical nature. Hallucinations are not uncommon in hysterics—usually hallucinations of sight, of terrifying nature, and often recognised as false. Paranoiac ideas are common and the insane jealousy of the hysteric comes under this head. The emotional variations are of the greatest importance: at one

extreme is the condition of overwhelming terror, sometimes accompanied by hallucinations and illusions, sometimes leading to suicidal or homicidal attacks. Alcohol or excitement may bring about a condition of wild fury, ending sometimes with deep exhaustion. Various confusional states are seen in hysteria: day-dreaming, somnambulism, and stuporose conditions come under this head. The most typical mental disturbance of hysterics is their characteristic delirium, usually of depressed emotional colour.

The hysterical psychoses not only develop out of the above elements, but are composed of a continuous series of the same, thus causing a very varying symptomatic picture. The psychosis may be of melancholic type or of a more chronic paranoiac type.

A third type where excitement varies with stuporose intervals is difficult to distinguish from certain conditions beginning at puberty and ending in weak-mindedness. For the diagnosis the origin in relation to external causes is important: the phenomena are superficial, the behaviour is theatrical, the patient seeks an audience. The prognosis is uncertain: recovery is possible after years, relapses are to be feared. The acute depressed form is more hopeful than the paranoiac form.

C. MACFIE CAMPBELL.

**THE PSYCHO-EPILEPSY OF JANET AND THE THEORY OF**  
(284) **STORCH AND FOERSTER.** DE BUCK, *Journ. de Neurol.*,  
May 5, 1904.

DR DE BUCK gives a description of Janet's theory of the pathogenesis of psycho-epilepsy, according to which psycho-epilepsy and psychæsthenia depend on the enfeeblement of superior psychological functions, particularly those concerned in the feeling of reality of the external world and our relations to it (*sentiment de complétude, sentiment ou fonction du réel*). Personality being a synthesis, a continuous process in course of evolution, the psychological function subserving this feeling, i.e. the continuous adaptation of mental processes and acts to the environment, must involve a psychological tension proportional to the richness of the synthesis, a complexity of association of images and ideas. There is then for Janet a psychological hierarchy, and if the rate or sequence in which the psychological functions are lost be considered, it is apparent that the loss is in direct relation to the position in this hierarchy, of what Spencer termed the coefficient of reality; the higher the coefficient, the greater the complexity, the less stable it is and the earlier to be affected in morbid mental states.

Psycho-epilepsy and psychæsthenia—for Janet corresponding states, one acute, the other chronic—rest on a failure of psychological

tension, a lowering of mental level, to which the phobias, obsession, tics, etc., present in these cases, are secondary and derived.

Dr de Buck criticises this theory and maintains that Janet, in deriving all disturbance of this feeling of reality from a general lowering of mental level, leaves out of count essential factors, and, following Wernicke, Storch and Foerster, shows that perception is composed of two essential elements, (1) organic and (2) purely sensorial. Storch's interesting and well-known theory of the somato-psyche and the pacho- or allopsyche is then carefully explained, in so far as it concerns the subject under discussion. The cases Janet had quoted are then retroversed and examined in the light of the Storch-Foerster theory, and the author gives his reasons for classing as somato-psychoses a series of cases Janet classes as psycho-epilepsy and psychæsthenia due to a lowering of mental level. He is of opinion that Janet errs by including under one rubric both perceptive and apperceptive reality, and holds that whilst the theory of a diminution of psychological tension explains disturbances of the latter occurring in many psychæsthenics, diminution or loss of perceptive reality is best elucidated by the Storch-Foerster theory.

R. CUNYNGHAM BROWN.

## TREATMENT.

**EXERCISE TREATMENT IN PARALYSIS AGITANS.** R. FRIED-  
(285) LAENDER, *Ztschr. f. diätet. u. physikal. Therapie*, Bd. 7, H. 12,  
March 1904.

THE author describes a method of exercise treatment which he thinks will be of service in cases of paralysis agitans.

Exercises producing fatigue are contra-indicated in this disease, since the rigidity and tremor are increased thereby, and the general condition is made worse. All the exercises should be of short duration, and should be separated by intervals of rest.

The hypertonus of the muscles can be diminished by passive movements, carried out cautiously, slowly, and lightly. Rapid and strong passive movements cause pain in the muscles and joints, and may increase the rigidity and tremor.

Passive movements should be commenced in the fingers, hands, toes, and feet, and then gradually the larger muscle groups should be treated. At each joint 5 to 10 passive movements should be made in the various directions—at first to a slight extent, then gradually to the full extent. During these movements the patients should endeavour to relax the muscles. The part of the limb not submitted to passive movements should be fixed by the left hand of the medical man or nurse who is carrying out the treatment. As the rigidity is chiefly on the flexor muscles, passive movements

should be made in the direction of extension. Under this treatment the muscles become more elastic, and passive and active movements can be carried out more easily. The muscular rigidity and tremor diminish. At first the effect is only temporary; but in time a permanent diminution of the rigidity and tremor occurs. Even in severe cases of paralysis agitans the author has obtained a certain amount of improvement.

A mild massage treatment of the skin and muscles may be combined with the passive movements.

In suitable cases active exercises may be also carried out.

When the disease is not too advanced, the patient is able to voluntarily control the tremor for a short time. Also the abnormal position of the limbs and trunk can be voluntarily corrected for a short time. By extra exertion and concentration of the attention, definite movements, which are otherwise performed slowly and with difficulty, can be performed more rapidly and with greater elasticity. This power of voluntarily influencing the tremor and abnormal attitude is only temporary, but it may be increased and educated to a certain extent by strong cerebral impulses.

The cases for the treatment should not be too advanced. When the disease is so advanced that the patient is no longer able to voluntarily influence the tremor and muscular rigidity, the exercise treatment is useless. But if the patient has still the power of influencing the tremor and rigidity, even to a slight degree, this power can be increased by practice. The following is the method suggested by Friedlaender:—

By effort and concentration of attention, the patient endeavours to suppress the tremor in the hands or feet. If this can be done even for a short time, by practice the limb can be held quiet for longer periods, and finally it can be held quiet without any great effort.

The abnormal flexed position of the head, trunk and limbs should be explained to the patient. By effort and by attention he should endeavour to correct this abnormal attitude, first in the sitting position, later in the standing posture. This is carried out by voluntary or active contractions of the extensor muscles. With the active contractions of the extensor muscles, normally there is simultaneously an inhibition of the innervation of the flexors, and hence such exercises diminish the muscular rigidity of the flexors in paralysis agitans. By the use of suitable apparatus, slight resistance can be made to the action of the extensor muscles, and this increases the effect of the treatment; but the resistance should only be slight. The movements ought, at the most, to be carried out ten times. Frequent pauses are desirable—best after each exercise. The pulse should be watched, and if it becomes



rapid the exercises should be discontinued until the pulse frequency returns to the normal.

Between these exercises the patient should practise walking. When walking he should endeavour to raise the head and straighten the vertebral column; and the eyes should be fixed on some object at a level above the height of the body. The patient should endeavour to correct the flexed position of the arms, and should swing them in the normal manner in walking.

To correct the abnormal gait three points are of importance: (1) the extension of the trunk and vertebral column, especially at the moment when the advancing leg is placed on the ground and becomes the supporting limb; (2) the gradual spring (or "unfolding") of the foot; (3) the practising of slow walking. Every time when the advancing leg is placed on the ground a short pause should be made and the trunk straightened. After a short time the patient should also endeavour to walk with longer steps.

Patients who have a tendency to propulsion should practise walking backwards, since in this exercise there is a tendency to straighten the back. Other movements should be practised, such as standing up and sitting down, going up and down stairs. Various methods of treatment of the hand muscles should be carried out, such as holding the fingers still, systematic extension movements, and exercises with Frenkel's stopper board or Frenkel's ball apparatus, in order that the patient may practise rapid and energetic contraction of the arm muscles.

Briefly stated, the exercise treatment advocated by Friedlander consists of—

1. Passive exercises, especially extension movements.
2. Active exercise of the extensor muscles, finally in opposition to slight resistance.
3. Exercise treatment in the strict sense, especially walking exercises, in which through attention and voluntary effort the deformities of position are corrected and propulsion and other abnormalities in movement counteracted.
4. Special exercise treatment of the upper limbs, active and passive extension movements, finer movements with apparatus, etc.

Friedlander admits that his experience is, as yet, too small to enable him to form a fair estimate of the value of the exercise treatment in paralysis agitans. But he is decidedly of opinion, that in suitable cases there is a prospect of good results, and, at all events, of an improvement of the motor symptoms. Of course, too much must not be expected from the treatment: neither a cure of the disease nor complete relief of the characteristic motor symptoms can be obtained thereby.

R. T. WILLIAMSON.

## Review

**DES SCLÉROSES COMBINÉES DE LA MOËLLE** Docteur O. CROUZON. G. Steinheil, Paris, 1904.

THIS valuable thesis for the Doctorate of Medicine was undertaken at the suggestion of Professor Pierre Marie, and it affords a very full consideration of the clinical, pathological, and etiological aspects, as far as our present knowledge goes, of the acquired forms of combined sclerosis. These are divided into five groups:— (1) Those associated with tabes; (2) with general paralysis; (3) with the various affections in which there is spasticity of the lower limbs; (4) the combined sclerosis of old people; and (5) the lateral sub-acute combined sclerosis occurring in anæmias, intoxications, and in the various cachexias.

The characteristic features of combined tabetic sclerosis are shown to be a gait with trailing of the lower limbs, paraplegia, and Babinski's sign, with certain accessory signs not always present, such as Strümpell's phenomenon and cramp-like pains. This form of combined sclerosis occurs apparently in a proportion of 1 to 15 of ordinary tabes, and is attributed to the spread of the sclerotic process from the posterior columns and their membranes to the lateral columns, due to an overflow of the inflammatory processes from the lymphatic system of the posterior columns into that of the lateral.

The diagnosis of the combined sclerosis of general paralysis is at present as difficult as that of the ordinary tabetic form is simple and exact.

In spasmodic combined sclerosis C. describes the following varieties:—

1. One in which the signs of ordinary tabes and spasmodic paraplegia are associated.
2. The paretico-amyotrophic form of Pal and Kattwinkel.
3. The ataxic paraplegia of Gowers.
4. The spasmodic ataxo-cerebellar type.
5. The type of spasmodic paraplegia of Dejerine and Sottas, in which an undiagnosable lesion of the posterior columns is associated with that of the lateral columns.
6. Spasmodic syphilitic paraplegia.

Crouzon enters very fully into the differential diagnosis of these various forms.

The varieties and diagnostic features of the arterio-sclerosis of old people are next considered, and the cause is regarded as a sclerosis of the vessels of the posterior columns, especially of the artery of the posterior median septum.

The sub-acute combined sclerosis, which appear to be more infrequent in France than in Britain, are fully considered, especially with reference to the forms described by Risien Russell, and the very frequent relation of these forms to disseminated sclerosis or myelitis.

The thesis concludes with the discussion of the pathogeny of the various forms of combined sclerosis which in the majority of cases are regarded as pseudo-systematic, and depending mainly upon lesions of the vascular and lymphatic systems.

ALEXANDER BRUCE.

## Bibliography

### ANATOMY

- MOURRE. Sur la variation des corpuscules de Nissl dans diverses conditions physiologiques. *Soc. de Biol.*, juin 10, 1904, p. 907.
- TRICOMI-ALLEGRI. I calici di Held nei centri acustici. *Nervaze*, Vol. vi., f. 2, 1904, p. 155.
- SERGE SOUKHANOFF. Contribution à l'étude de l'aspect externe des prolongements protoplasmiques des cellules nerveuses colorés par le bleu de méthylène. *Nervaze*, Vol. vi., f. 2, 1904, p. 171.
- L. FUSSEP. Ueber die Assoziationsfasern der feinkörnigen Schicht der Kleinhirnrinde. *Neurol. Centralbl.*, Juli 16, 1904, S. 655.
- VAN GEHUCHTEN. Boutons terminaux et réseau péricellulaire. *Nervaze*, Vol. vi., f. 2, 1904, p. 217.
- ALBERT MICHOTTE. La fibre nerveuse et sa bifurcation dans les ganglions (méthode de Cajal). *Nervaze*, Vol. vi., f. 2, 1904, p. 201.
- VAN GEHUCHTEN. Contribution à l'étude des voies olfactives. *Nervaze*, Vol. vi., f. 2, 1904, p. 191.
- VAN GEHUCHTEN. Le corps restiforme et les connexions bulbo-cérébelleuses. *Nervaze*, Vol. ix., f. 2, 1904, p. 123.
- CHARLES E. INGBERT. An Enumeration of the Medullated Nerve Fibres in the Ventral Roots of the Spinal Nerves of Man. *Journ. Comp. Neurol. and Psychol.*, June 1904, p. 209.
- G. STERZI. Die Blutgefäße des Rückenmarks. Bergmann, Wiesbaden, 1904.
- IRVING HARDESTY. On the Development and Nature of the Neuroglia. *Am. Journ. Anat.*, Vol. iii., No. 3, 1904, p. 229.
- O. CHARNOCK BRADLEY. The Mammalian Cerebellum, its Lobes and Fissures. *Journ. Anat. and Physiol.*, July 1904, p. 443.
- EDWARD A. SPITZKA. Hereditary Resemblances in the Brains of Three Brothers. *Am. Anthropologist*, April-June 1904, p. 307.
- WILFRED HARRIS. The True Form of the Brachial Plexus and its Motor Distribution. *Journ. Anat. and Physiol.*, July 1904, p. 399.
- E. ZUCKERKANDL. Zur Morphologie des Affengehirns. *Ztschr. f. Morphol. u. Anthropol.*, Bd. 7, H. 2, 1904, S. 223.
- J. DRÄSEKE. Zur Kenntnis des Rückenmarks und der Pyramidenbahnen von Talpa europaea. *Monats. f. Psychiat. u. Neurol.*, Juni 1904, S. 401.
- S. R. CAJÁL. Ueber einige Methoden der Silberimprägnierung zur Untersuchung der Neurofibrillen, der Achseneylinder und der Endverzweigungen. *Ztschr. f. wiss. Mikrop.*, Juli 1904, S. 401.
- W. FORD ROBERTSON. A Method of producing a Selective Deposit of Silver in Blocks of Tissue and of obtaining Sections suitable for Toning with Gold, Platinum and other Metals. *Rev. Neurol. and Psychiat.*, July 1904, p. 522.
- M. V. LENHOSSEK. Ramón y Cajals neue Fibrillenmethode. *Neurol. Centralbl.*, Juli 1, 1904, S. 593.

## PHYSIOLOGY

- MDLLE. IOTAYKO. La dualité fonctionnelle du muscle. *Journ. de Neurol.*, juin 20, 1904, p. 221.
- L. J. J. MUSKENS. Studies on the Maintenance of the Equilibrium of Motion and its Disturbances, so-called "Forced Movements." *Journ. Physiol.*, June 30, 1904, p. 204.
- DENIS COURTADE. Rapport entre la contraction volontaire et les réactions électriques neuro-musculaires. *Ann. d'Électrobiol. et de Radiol.*, T. vii., No. 1, 1904, p. 15.
- CLUZET. Sur l'excitation des nerfs par décharges de condensateurs. *Ann. d'Électrobiol. et de Radiol.*, No. 6, 1904, p. 697.
- IOTAYKO. Mécanisme physiologique de la réaction de dégénérescence des muscles. *Ann. d'Électrobiol. et de Radiol.*, No. 6, 1904, p. 677.
- CLUZET. Étude comparative des manifestations électrotoniques des nerfs et de l'inversion de la loi des secousses. *Ann. d'Électrobiol. et de Radiol.*, No. 6, 1904, p. 661.
- DESSY et GRANDIS. Contribution à l'étude de la fatigue. Action de l'adrénalin sur la fonction du muscle. *Arch. Ital. de Biol.*, Vol. xii., f. ii., 1904, p. 225.
- A. MOSSO. Théorie de la tonicité musculaire basée sur la double innervation des muscles striés. *Arch. Ital. de Biol.*, Vol. xii., f. ii., 1904, p. 183.
- A. MOSSO. Réponse au Prof. Fanu, à propos des observations critiques relatives à la note "Théorie de la tonicité," etc. *Arch. Ital. de Biol.*, Vol. xii., f. ii., 1904, p. 381.
- PATRIZI. Un instrument (ergostéthographe) pour mesurer la fatigue des muscles respirateurs chez l'homme. *Arch. Ital. de Biol.*, Vol. xii., f. ii., 1904, p. 215.
- R. S. WOODWORTH and C. S. SHERRINGTON. A Pseudoaffective Reflex and its Spinal Path. *Journ. Physiol.*, June 30, 1904, p. 234.
- G. DURANTE. A propos de la théorie du neurone. Terminaisons fibrillaires. Régénération autogène; différenciation fonctionnelle et rôle du cylindrax. Sensibilité récurrente et suppléances sensitive. Propagations des dégénérescences. *Rev. Neurol.*, juin 30, 1904, p. 573.
- J. N. LANGLEY. On the Question of Commissural Fibres between Nerve-cells having the same Function and situated in the same Sympathetic Ganglion, and on the Function of Post-ganglionic Nerve Plexuses. *Journ. Physiol.*, June 30, 1904, p. 244.
- C. L. HERRICK. Color Vision. *Journ. Comp. Neurol. and Psychol.*, June 1904, p. 274.
- A. PICK. Ueber compensatorische Vorgänge im menschlichen Rückenmark. *Neurol. Centralbl.*, Juli 16, 1904, S. 641.
- W. PAGE MAY. The Innervation of the Sphincters and Musculature of the Stomach. *Journ. Physiol.*, June 30, 1904, p. 260.
- L. V. FRANKL-HOCHWART und ALFRED FRÖHLICH. Ueber die corticale Innervation der Harnblase. *Neurol. Centralbl.*, Juli 16, 1904, S. 646.
- PAUL SCHULTZ. Die Beteiligung des Sympathicus an der Kehlkopfnnervation. *Arch. f. Laryngol. u. Rhinol.*, Bd. 16, H. 1, 1904, S. 1.
- U. SCHEVEN. Ueber die Wiederkkehr der elektrischen Erregbarkeit des Gehirns nach temporärer Anämie. *Arch. f. Psychiat.*, Bd. 88, H. 3, 1904, S. 928.
- G. JELGASMA. De physiologische betekenenis van het Cerebellum. *Scheitma & Kolkema*, Amsterdam, 1904.
- MAX ROTHMANN. Ueber experimentelle Läsionen des Centralnervensystems beim anthropomorphen Affen. *Arch. f. Psychiat.*, Bd. 88, H. 3, 1904, S. 1020.

## PSYCHOLOGY

- F. BRUPBACHER. Die Psychologie der Dekadenten. H. Thurov, Zürich, 1904.
- BINET. L'imagination littéraire. *L'Année Psychologique*, T. x., 1904.
- LÉCAILLON. La biologie et la psychologie d'une araignée. *L'Année Psychologique*, T. x., 1904.
- LARGUIER DES BANCELS. Expériences sur la mémoire. *L'Année Psychologique*, T. x., 1904.
- BINET. Questions de technique céphalométrique. *L'Année Psychologique*, T. x., 1904.
- BINET. Sommaire des travaux en cours à la Société de Psychologie de l'enfant. *L'Année Psychologique*, T. x., 1904.

- ZWAARDEMAKER. Expériences sur les limites de l'audition. *L'Année Psychologique*, T. x., 1904.  
 TOULOUSE, VASCHIDE, et PIÉRON. Technique de Psychologie expérimentale (examen des sujets). Octave Doin, Paris, 1904, 4 fr.  
 G. F. LIPPS. Die Massmethoden der experimentellen Psychologie. *Arch. f. d. ges. Psychol.*, Bd. 3, H. 2, 1904, S. 153.  
 FRIEDRICH CONTRAT. Hermann von Helmholtz' psychologische Anschauungen. Max Niemeyer, Halle, 1904.

### PATHOLOGY

- O. V. LEONOWA--V. LANGE. Zur pathologischen Entwicklung des Centralnervensystems. *Arch. f. Psychiat.*, Bd. 38, H. 3, 1904, S. 862.  
 BORST. Neue Experimente zur Frage nach der Regenerationsfähigkeit der Gehirns. *Beit. zur pathol. Anat. u. allg. Pathol.*, Bd. 36, H. 1, 1904, S. 1.  
 BIRCH-HIRSCHFELD. Die Nervenzellen der Netzhaut unter physiologischen und pathologischen Verhältnissen mit besonderer Berücksichtigung der Blindung. *München. med. Wchnschr.*, Juli 5, 1904, S. 1192.  
 E. LUGARO. Sur la pathologie des cellules des ganglions sensitifs. *Arch. Ital. de Biol.*, Vol. xli, f. 2, 1904, p. 201.  
 MOURRE. Modifications structurales des cellules nerveuses consécutives à l'administration de quelques substances toxiques. *Soc. de biol.*, juin 10, 1904, p. 909.  
 BURZIO. Sulle alterazioni delle fibre nervose spinali e dei gangli intervertebrali in alcune forme di psicosi croniche. *Ann. di Freniat.*, Vol. xiv, f. 2, 1904, p. 97.  
 M. CHIO. Sur quelques particularités de structure de la fibre nerveuse myélinique soumise à l'action de l'acide osmique. *Arch. Ital. de Biol.*, Vol. xli, f. 2, 1904, p. 277.  
 DAVID. Les lésions expérimentales du système nerveux sympathique influentes sur la pénétration de la toxine diphtérique et sur la résistance organique? Ducourtieux et Goût, Limoges, 1904.  
 RIGHETTI. Contributo allo studio dell'anencefalia e dell'amielia. *Riv. di Patolog. nerv. e ment.*, Vol. ix, f. 6, 1904, p. 257.  
 PAUL BONCOUR. Modifications crâniennes consécutives aux atrophies cérébrales unilatérales. *Arch. de neurol.*, juillet 1904, p. 28.  
 DELAFOSSE. Névromes plexiformes, ou Névromes racémeux. Thèse. Morel, Lille, 1904.

### CLINICAL NEUROLOGY AND PSYCHIATRY

#### GENERAL—

- C. FÜRSTNER. Neuropathologie und Psychiatrie. *Arch. f. Psychiat.*, Bd. 38, H. 3, 1904, S. 895.  
 JUDSON S. BURY. Trauma in Relation to Disease of the Nervous System. Sheratt & Hughes, Manchester, 1904.  
 VERAGUTH. Kultur und Nervensystem. Schulthees & Co., Zürich, 1904, M. 1.  
 GEORGE OGILVY. What is Syphilitic Disease of the Nervous System? Medical Publishing Co., Ltd., London, 1904.  
 RUDOLF VON HÖSSLIN. Die Schwangerschaftslähmungen der Mütter. *Arch. f. Psychiat.*, Bd. 38, H. 3, 1904, S. 780.  
 BÉZY. Paralysie et Fièvre typhoïde chez l'enfant. Marquès et Cie, Toulouse, 1904.  
 HOORWEG und ZIEHEN. Elektrodiagnostische Untersuchungen mit Hilfe der Kondensatormethode. *Monatsschr. f. Psychiat. u. Neurol.*, Juni 1904, S. 426.

#### MUSCLES—

- VON HÖSSLIN. Juvenile Muskeldystrophie bei einem älteren Manne nach Trauma. *München. med. Wchnschr.*, Juni 23, 1904, S. 1156.

#### PERIPHERAL NERVES—

- CAMPBELL DYKES. An Outbreak of Beri-beri in an Assam Jail. *Indian Med. Gaz.*, July 1904, p. 201.  
 F. W. PAVY. On Diabetic Neuritis. *Lancet*, July 2, 1904, p. 17.  
 THIELLE. Sciatiques aiguës et subaiguës traitées par les courants continus et la franklinisation. *Bull. Soc. Française d'Electrother. et de Radiol.*, mai 1904, p. 127.  
 JOHN A. WYETH. Surgical Lesions of the Axillary Plexus. *Journ. Am. Med. Assoc.*, June 25, 1904, p. 1872.  
 SCHULTZ. Ueber Fasallähmung, speziell Peroneallähmung, bei Rübenarbeitern. *Deutsche Arch. f. klin. Med.*, Bd. 80, H. 5-6, 1904, S. 520.

- JAMES HENDRIE LLOYD.** Two Cases of Paralysis of the Ulnar Nerve, one following a severe attack of Typhoid Fever, the other caused by Pressure during Occupation. *Med. News*, June 18, 1904, p. 1156.
- Herpes Zoster.**—CARLO QUADRONE. Herpes zoster nel dominio delle branche cervicali inferiori e delle branche toraciche superiori a topografia radicolare e ad intensa reazione meningea. *Riv. di Patolog. nerv. e ment.*, Vol. ix., f. 7, 1904, p. 333.

# SPINAL CORD—

- LAPINSKY.** Über die Lokalisation motorischer Funktionen im Rückenmark. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 26, H. 4-6, 1904, S. 457.
- Tabes.**—PERRERO EMILIO. Le forme fruste della tabe spinale e loro etiologia. Torino, 1904.
- BREGMAN.** Über Grün- und Violettsehen bei Tabes dorsalis. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 26, H. 4-6, 1904, S. 525.
- BERNARDICOU.** Contribution à l'étude des Rapports symptomatiques entre le Tabes et l'Hystérie. *Thèses de Paris*, 1904.
- E. COESTER.** Zur Diagnose und Therapie des Tabes. *Berl. Min. Wchnschr.*, Juli 4 u. 11, 1904, S. 782.
- M. FAURE.** Pronostic actuel et thérapeutique du tabes. *Gaz. méd. de Nantes*, juin 4, 1904, p. 445.
- C. L. DANA.** Special Exercises for Tabes Dorsalis. *Pub. of Cornell Univ. Med. Coll.*, Vol. i., 1904.
- Disseminated Sclerosis.**—MÜLLER. Die multiple Sklerose des Gehirns und Rückenmarks. G. Fischer, Jena, 1904, M. 10.
- BYROM BRAMWELL.** Lecture on Disseminated Sclerosis. *Clin. Journ.*, June 22, 1904, p. 148.
- SMITH ELY JELIFFE.** Multiple Sclerosis: Its Occurrence and Etiology. *Journ. Nerv. and Ment. Dis.*, July 1904, p. 446.
- W. G. SPILLER and C. D. CAMP.** Multiple Sclerosis, with a Report of Two Additional Cases, with Necropsy. *Journ. Nerv. and Ment. Dis.*, July 1904, p. 433.
- THOMAS BUZZARD.** A Clinical Lecture on Remissions and Relapses in Insular Sclerosis. *Lancet*, July 16, 1904, p. 131.
- A. F. TREDGOLD.** Disseminated Sclerosis. An Account of the Microscopical Examination of Three Cases, with some Observations on the Pathogenesis of the Disease. *Rev. Neurol. and Psychiat.*, July 1904, p. 497.
- Myelitis.**—SCHMAUS. Die Anwendung des Entzündungsbegriffes auf die Myelitis. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 26, H. 4-6, 1904, S. 390.
- Pressure Paraplegia.**—J. RAMSEY HUNT. A Case of Mid-dorsal Pott's Disease, Compression-Myelitis, Recovery; and a Study of Gowars' Tract. *Pub. of Cornell Univ. Med. Coll.*, Vol. i., 1904.
- H. JUDET et BAUDOUIN.** Mal de Pott et déambulation. *Arch. gén. de méd.*, juillet 5, 1904, p. 1672.
- Tumours.**—A. TURNER. Diagnosis of Tumours of the Spinal Cord. *Clin. Journ.*, June 8, 1904, p. 122.
- HARVEY CUSHING.** Intradural Tumour of the Cervical Meninges. *Ann. of Surg.*, June 1904, p. 834.
- Poliomyelitis Anterior Chronica.**—AOYAMA. Über einen Fall von Poliomyelitis anterior chronica mit Sektionsbefund. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 26, H. 4-6, 1904, S. 375.
- Syringomyelia.**—L. DOJMI DI DELUPIS. Lepra oder Syringomyelie? *Wien. med. Wchnschr.*, Juni 4, 1904, S. 1068.
- H. J. VETLESEN and FRANCIS HARLITZ.** Kasuistische Beiträge zur Lehre von der Syringomyelie. *Nord. med. Arch.*, Abt. II., H. 1, 1904, S. 61.
- TEDESKO.** Über Knochentrophie bei Syringomyelie. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 26, H. 4-6, 1904, S. 336.
- Neuratomyelia.**—SCHOTT. Ein Fall von traumatisch entstandener Hämatomyelia. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 26, H. 4-6, 1904, S. 457.
- Contusion.**—P. STOLPER. Zur Lehre von der Rückenmarkerschütterung. *Vierteiljahrsschr. f. gericht. Med.*, Juli 1904, S. 57.
- Cauda Equina.**—THEODORE H. WEISENBERG. A Clinical Report of Three Cases of Injury to the Lower Spinal Cord and Cauda Equina. *Am. Journ. Med. Sc.*, May 1904.
- Spinal Column.**—P. FIORENTINI. Sulle anchilosi dell'acolonna vertebrale con speciale riguardo alla spondilosi rizomelica. *Ric. crit. di clin. Med.*, Luglio, 1904, p. 429.
- ROBERT.** La Scoliose tuberculeuse. Kreis, Nancy, 1904.
- Lumbar Puncture.**—SCHLESINGER. Zytologische Untersuchungen des Liquor cerebrospinalis. *Deutsche med. Wchnschr.*, Juli 7, 1904, S. 1022.

DONATH. Der Phosphorsäuregehalt der Cerebrospinalflüssigkeit bei verschiedenen insbesondere Nervenkrankheiten. *Hoppe-Seyler's Ztschr. f. physiol. Chemie*, Bd. 42, H. 1-2, 1904, S. 141.

LEFAS. Hématologie et cytologie cliniques. Baillière et fils, Paris, 1904, 3 fr.

CHAUFFARD et BOLDIN. Un an de ponctions lombaires dans un service hospitalier. *Gaz. des Hôp.*, juin 28, 1904, p. 725.

#### BRAIN AND MEMBRANES—

**Meningitis.**—M. MICHEL et R. GAULTIER. Méningite tuberculeuse en plaques au cours d'une phthisie pulmonaire. *Arch. gén. de méd.*, juillet 5, 1904, p. 1878.

BRAIM. Pachymeningitis externa fungosa. *Vierteljahrsschr. f. gericht. Med.*, Juli 1904, S. 71.

CURSCHMANN. Ueber postreumatische Meningitis. *Deutsche med. Wchnschr.*, Juli 14, 1904, S. 1054.

CHARLES BROOK. A Case of Mania from Traumatic Meningitis; Trephining; Evacuation of Serous Effusion; Complete Recovery. *Lancet*, June 25, 1904, p. 1794.

**Hemorrhage.**—J. RAMSEY HUNT. A Case of Juvenile Apoplexy, with Autopsy. *Pub. of Cornell Univ. Med. Coll.*, Vol. i, 1904.

**Hemiplegia.**—T. H. WEISENBURG. Uremic Hemiplegia, with Changes in the Nerve Cells of the Brain and Cord. *Journ. Nerv. and Ment. Dis.*, July 1904, p. 456.

T. R. BRADSHAW. A Clinical Lecture on Two Cases of Hemiplegia. *Lancet*, July 16, 1904, p. 184.

**Encephalitis.**—W. SPIELMEYER. Zur Symptomatologie der Grosshirnencephalitis, speziell über eine epileptische Form dieser Erkrankung. *Centralbl. f. Nerv. u. Psychiat.*, Juni 1904, S. 371.

**Bulbar Paralysis.**—VON VOSS. Ein Fall von progressiver Bulbärparalyse. *St. Petersburg. med. Wchnschr.*, Juli 2, 1904, S. 267.

WILFRED HARRIS. Post-diphtheritic Chronic Bulbar Paralysis. *Lancet*, July 23, 1904, p. 209.

LEONARD K. HIRSCHBERG. Pseudobulbar (Glossopharyngo-Labial) Paralysis. *Med. News*, July 9, 1904, p. 68.

**Tumour.**—CHAS. W. HITCHCOCK. Tumors, Cranial and Intracranial. *Detroit Med. Journ.*, May 1904.

CAMPBELL MEYERS. A Case of Cerebral Tumor probably of Left Frontal Lobe. *Canadian Journ. of Med. and Sur.*, May 1904.

KUFFS. Ueber einen Fall von secundärem Carcinom der Brücke und des rechten Schläfenlappens bei primärem Uteruscarcinom. *Arch. f. Psychiat.*, Bd. 38, H. 3, 1904, S. 908.

J. RAMSEY HUNT. A Case of Tumor of the Right Frontal Lobe, with Symptoms simulating Paresis. *Pub. of Cornell Univ. Med. Coll.*, Vol. i, 1904.

**Abscess.**—C. H. R. JORDAN. Report of a Fatal Case of Multiple Otitic Abscesses in the Temporal Lobe, with Remarks. *Arch. of Otol.*, June 1904, p. 186.

**Syphilis.**—F. W. MOTT. Syphilitic Disease of the Brain. *Practitioner*, July 1904, p. 55.

DEMETRIO GALATTI. Klinischer Beitrag zum Hydrocephalus syphilitischen Ursprungs. *Wien. klin. Wchnschr.*, Juni 23, 1904, S. 705.

**Diplegia.**—HANS WACHSMUTH. Beiträge zur cerebralen Kinderlähmung. *Arch. f. Psychiat.*, Bd. 38, H. 3, 1904, S. 713.

**General Paralysis.**—SERRIGNY. La paralysie générale dans la Suisse romande (suite et fin). *Ann. méd.-psychol.*, juillet-août 1904, p. 48.

JOSEF A. HIRSCHL. Die Frühdiagnose der progressiven Paralyse. *Wien. med. Presse*, Juli 3 u. 10, 1904, S. 1820 u. 1870.

KARL ABRAHAM. Ueber einige seitens Zustandsbilder bei progressiven Paralyse. Apraxie, transkortikale sensorische Aphasie sensorisch-motorische Asymbole. *Allg. Ztschr. f. Psychiat.*, Bd. 61, H. 4, 1904, S. 502.

JEANTY. Lésions du cervelet chez les paralytiques généraux et les déments. Thèse. Ray, Lyon, 1904.

A. EITELBERG. Ohrensausen bei progressiver Paralyse. *Wien. med. Presse*, Juli 17, 1904, S. 1416.

#### GENERAL AND FUNCTIONAL DISEASES—

LEONARD GUTHRIE. On Neurotic Children. *Clin. Journ.*, June 29, 1904, p. 167.

FRANCIS HARE. Mechanism of the Paroxysmal Neuroses. W. E. Smith, Sydney, 1904.

- G. HUDSON-MAKUEN. Neuroses of Nasal Origin. *Am. Medicine*, June 4, 1904.
- Epilepsy.**—A. F. TREDGOLD. On the Relations of Epilepsy to Amentia. *Brit. Journ. Children's Dis.*, July 1904, p. 291.
- LACHMUND. Ueber vereinzelt auftretende Halluzinationen bei Epileptikern. *Monatschr. f. Psychiat. u. Neurol.*, Juni 1904, S. 434.
- GUSTAVE GOUIN. Étude de l'épilepsie expérimentale par les courants intermittents de basse tension. *Ann. d'Electrobiol. et de Radiol.*, T. vii, No 1, 1904, p. 37.
- C. L. DANA. The Formal Treatment of Epilepsy. *Pub. of Cornell Univ. Med. Coll.*, Vol. i, 1904.
- KONRAD ALT. Die diätetische Behandlung der Epileptiker in Vergangenheit und Gegenwart. *Ztschr. f. klin. Med.*, Bd. 35, 1904, S. 380.
- Paralysis Agitans.**—C. L. DANA. Special Exercises for Parkinson's Disease. *Pub. of Cornell Univ. Med. Coll.*, Vol. i, 1904.
- Chorea.**—FRANK R. FRY. Chorea and Graves' Disease. *Med. Fortnightly*, St Louis, May 10, 1904.
- Neurasthenia.**—MIECAMP. Pathogénie de la céphalée neurasthénique. *Arch. gén. de méd.*, juin 28, 1904, p. 1601.
- Hysteria.**—GUY L. CONNOR. Hysteria: Certain Manifestations. *Journ. of Michigan State Med. Soc.*, June 1904.
- DEBOVE. Pouls lent permanent et hystéro-traumatisme. *Presse méd.*, juillet 13, 1904, p. 441.
- HASSLAUER. Ueber hysterische Stimmstörungen. Stuber, Würzburg, 1904, M.—75.
- Neuralgia.**—J. NICOLAS. Les Névralgies des tuberculeux, des asthmatiques et des emphysemateux. Clermont-Ferrand, Mont-Louis, 1904.
- THIELLE. Névralgie tri-faciale droite, traitées par les courants à états variables. *Ann. d'Electrobiol. et de Radiol.*, T. vii, No. 1, 1904, p. 76.
- Tetany.**—SCHEIBER. Ein Fall von "atypischer" Tetanie mit anfänglichem Gesichtskrampf. *Wien. med. Wchnschr.*, Juni 25, 1904, S. 1206.
- JOSEF JACOBI. Struma und Tetani. *Wien. klin. Wchnschr.*, Juli 7, 1904, S. 768.
- RICHARTZ. Beitrag zur Lehre von der Tetanie bei Magenerweiterung. *Ztschr. f. klin. Med.*, Bd. 35, 1904, S. 369.
- HONIGMANN. Beitrag zur Symptomatologie des Magenkrebes und zum Vorkommen der Tetanie bei Magenkrankheiten. *Ztschr. f. klin. Med.*, Bd. 53, 1904, S. 347.
- VOSS. Zur Kasuistik der Tetanie mit myotonischen Erscheinungen. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 26, H. 4-6, 1904, S. 521.
- Tetanus.**—F. B. HOFMANN. Studien über den Tetanus. III. Zur Erklärung der scheinbaren Hemmungen am Nervmuskelpreparate. *Arch. f. d. ges. Physiol.*, Bd. 103, H. 7-8, 1904, S. 291.
- HAROLD COLLINSON. A Case of Traumatic Tetanus. *Lancet*, July 2, 1904, p. 23.
- The Prophylaxis and Treatment of Fourth of July Tetanus. Special Article. *Journ. Am. Med. Assoc.*, June 13, 1904, p. 1621.
- S. C. STANTON. The Prophylaxis of Tetanus. *Journ. Am. Med. Assoc.*, June 11, 1904, p. 1555.
- Torticollis.**—ARMIN STEYERTHAL und BERNH. SOLGER. Ueber Torticollis spasmodicus. *Arch. f. Psychiat.*, Bd. 33, H. 3, 1904, S. 949.
- Myasthenia Gravis.**—OPPENHEIM. Zur myasthenischen Paralyse. *Deutsche med. Wchnschr.*, Juli 14, 1904, S. 1053.
- Adiposis Dolorosa.**—J. HAMMOND. An Instance of Adiposis Dolorosa in Two Sisters. *Brit. Med. Journ.*, July 16, 1904, p. 121.

#### ALCOHOLISM, ETC.—

- FREDERIC C. COLEY. Some Points in the Etiology of Inebriety. *Brit. Journ. Inebriety*, July 1904, p. 22.
- ALZHEIMER. Das Delirium alcoholicum febrile Magnan's. *Centralbl. f. Nerven- u. Psychiat.*, Juli 15, 1904, S. 437.
- PICARD. Les auto-accusateurs alcooliques. Thèse. Roussel, Paris, 1904.
- MARNAY. La suggestion hypnotique dans la cure des buveurs d'habitude. Thèse de Paris, Jouve, 1904.
- STANLEY B. ATKINSON. The Forensic Psychology of Inebriety. *Brit. Journ. Inebriety*, July 1904, p. 16.



ROBERT JONES. The Relation of Inebriety to Mental Diseases. *Brit. Journ. Inebriety*, July 1904, p. 4.

P. T. HALD. Ein Fall von Veronalvergiftung. *Centralbl. f. Nervenheilk. u. Psychiat.*, Juni 1904, S. 369.

#### MENTAL DISEASES—

E. KRAEPELIN. Vergleichende Psychiatrie. *Centralbl. f. Nervenl. u. Psychiat.*, Juli 15, 1904, S. 433.

TH. ZIEHEN. Die Entwicklungsstadien der Psychiatrie. *Berl. klin. Wchnschr.*, Juli 18, 1904, S. 777.

RUDOLF GANTER. Untersuchungen auf Degenerationszeichen bei 251 geisteskranken Männern. *Arch. f. Psychiat.*, Bd. 38, H. 3, 1904, S. 978.

VIGOUROUX et JULEQUIER. La contagion mentale. Octave Doin, Paris, 1904, 4 fr.

LEROY. Idiotie myxoedémateuse. Charles Lavauzelle, Paris, 1904, 1 fr. 25.

L. RONCORONI. Nota sulla patogenesi delle allucinazioni. *Riv. di Patolog. nerv. e ment.*, Vol. ix., f. 7, 1904, p. 314.

E. TANZI. Sopra una teoria dell' allucinazione. *Riv. di Patolog. nerv. e ment.*, Vol. ix., f. 7, 1904, p. 322.

DE BUCK. Association chez un idiot de maladie de Raynaud et de gangrène névrotique cutanée multiple. *Bull. de la Soc. de Méd. ment. de Belgique*, N. 114, 1904.

A. HOCHÉ. Enthüllung und Benennung der Psychoosen mit Rücksicht auf die Anforderungen der ärztlichen Prüfung. *Arch. f. Psychiat.*, Bd. 38, H. 3, 1904, S. 1070.

SERGE SOUKHANOFF. Differential Diagnosis in Cases of Ideo-obsessional Constitution. *Journ. Ment. Pathol.*, Vol. v., Nos. 2-3, 1904, p. 57.

LOUIE G. ROBINOVITCH. Suicidal and Homicidal Acts. Their Clinical Aspects and Medico-legal Significance. *Journ. Ment. Pathol.*, Vol. v., Nos. 2-3, 1904, p. 71.

HOPPE. Simulation und Geistesstörung. *Vierteljahrschr. f. d. gericht. Med.*, Juli 1904, S. 91.

FR. GEIST. Tuberkulose und Irrenanstalten. *Allg. Ztschr. f. Psychiat.*, Bd. 61, H. 4, 1904, S. 477.

REICHARDT. Ueber akute Geistesstörungen nach Hirnerschütterung. *Allg. Ztschr. f. Psychiat.*, Bd. 61, H. 4, 1904, S. 524.

WEYGANDT. Über Beerdigungsatteste bei Selbstmördern. *Allg. Ztschr. f. Psychiat.*, Bd. 61, H. 4, 1904, S. 552.

DIEHL. Der Kopfschmerz beim manisch-depressiven Irresein. *Monatsschr. f. Psychiat. u. Neurol.*, Juni 1904, S. 419.

G. LOMER. Ein Fall von seniler Demenz mit gleichzeitiger genuiner Schrumpfnier. *Deutsche med. Wchnschr.*, Juli 1904, S. 952.

ROB. SOMMER. Kriminal Psychologie und strafrechtliche Psychopathologie. J. A. Barth, Leipzig, 1904.

#### SPECIAL SENSES AND CRANIAL NERVES—

TOMMASO PRODI. Gonoiastro bilaterale e scomparsa dei disturbi visivi in un caso d'acromegalia. *Gaz. d. ospedali e d. cliniche*, Giugno 12, 1904, p. 788.

FLATAU. Neuritis optica bei Paratyphus. *München. med. Wchnschr.*, Juli 12, 1904, S. 1245.

KAMPHERSTEIN. Beitrag zur Pathologie und Pathogenese der Stauungspapille. *Klin. Monatsbl. f. Augenheilk.*, Juni 1904, S. 501.

GRASSET. La déviation conjuguée des yeux et l'hémianopsie. *Rev. Neurol.*, juillet 15, 1904, p. 645.

H. GRADLE. Paralysis of the Associated Lateral Eye Movements, presumably due to Acute Poliomyelitis Superior, ending in Recovery. *Arch. of Ophthalm.*, May 1904, p. 268.

KLIEN. Über Inkoordination der Augenbewegungen nach einer oberflächlichen Gehirnläsion. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 26, H. 4-6, 1904, S. 327.

A. PAPADAKI. Paralysie du moteur oculaire commun chez un tabétique. Examen anatomique. *Rev. Neurol.*, juin 30, 1904, p. 585.

BERTOLOTI. Il segno d'Argyll e la meningite sifilitica. *Riv. crit. di clin. med.*, Giugno 4, 11 e 18, 1904.

MORTIMER FRANK. Hysterical Iridoplegia and Cycloplegia, with Report of a Case. *Arch. of Ophthalm.*, May 1904, p. 299.

L. E. O. ARQUIER. Considérations générales sur la suppléance nerveuse dans un cas de paralysie faciale. *Thèse de Bordeaux*, avril 1904.

FONCK. Klinischer Beitrag sur Kenntnis der motorischen Innervationsstörungen der Uvula. *Arch. f. Laryngol. u. Rhinol.*, Bd. 16, H. 1, 1904, S. 48.

## REFLEXES—

- FRIEDLÄNDER. Die Hautreflexe an den unteren Extremitäten unter normalen und pathologischen Verhältnissen. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 26, H. 4-6, 1904, S. 412.
- W. V. BECHTEREW. Ueber eine besonderen Bengeflex der Zehen. KURT MENDEL. Bemerkungen zu vorstehender Mittheilung. *Neurol. Centralbl.*, Juli 1, 1904, S. 609.
- DENIS G. ZESAS. Ueber den Babiniskischen Zehenreflexe. *Med. Blätter*, Juni 23, 1904, S. 303.
- HUTNER. Zur Diagnostik des pathologischen Rausches (Störungen der Reflexe). *Deutsche med. Wchnschr.*, Juli 14, 1904, S. 1057.

## MISCELLANEOUS SYMPTOMS—

- LUDWIG MANN. Zur Symptomatologie des Kleinhirns (über cerebellare Hemistaxie und ihre Entstehung). *Monatsschr. f. Psychiat. u. Neurol.*, Juni 1904, S. 409.
- SIEFERT. Ueber functionelle Hemisthetose. *Arch. f. Psychiat.*, Bd. 33, H. 3, 1904, S. 844.
- ROBERT M. DALEY. Primary Myokymia: with Report of a Case. *Med. News*, July 2, 1904, p. 12.
- OLIVA. Due casi di inversione sessuale. *Ann. di Freniat.*, Vol. xiv, f. 2, 1904, p. 138.
- HERMANN GUTZMANN. Die soziale Bedeutung der Sprachstörung. *Klin. Jahrb.*, Bd. 12, H. 3, 1904, S. 295.
- HERZOG. Casuistischer Beitrag zur Lehre von der motorischen Apraxia. *Ztschr. f. Klin. Med.*, Bd. 53, 1904, S. 332.
- BOURDON et DIDE. Un cas d'amnésie continue avec asymbolie tactile, compliqué d'autres troubles. *L'Année Psychologique*, T. x., 1904.
- DEJERINE et ANDRÉ THOMAS. Un cas de oscillé verbale avec agraphie suivi d'autopsie. *Rev. Neurol.*, juillet 15, 1904, p. 655.
- C. L. DANA. Special Exercises for Aphasia. *Pub. of Cornell Univ. Med. Coll.*, Vol. i., 1904.
- AUG. HEGAR. Der Stotterer vor dem Strafrichter. *Allg. Ztschr. f. Psychiat.*, Bd. 61, H. 4, 1904, S. 461.

## TREATMENT—

- DESCAMPS. Traitement des maladies nerveuses. Pariset, Paris, 1904.
- ANDRÉ THOMAS. Traitement de l'hystérie et de la neurasthénie par l'isolement et la psychothérapie. *Presse Méd.*, juillet 9, 1904, p. 434.
- SIEGMUND AUERBACH. Zur hydiatischen Behandlung der funktionellen Neurosen. *Ztschr. f. diät. u. physik. Therapie*, Juli 1904, S. 196.
- WEDENSKY. Ueber das Entstehen und die Natur der Nervennarkose. *Ztschr. f. Elektrother.*, Bd. 6, H. 6, 1904, S. 166.
- MELTZER. Die staatliche Schwachsinnigenfürsorge in Königreich Sachsen. II. *Allg. Ztschr. f. Psychiat.*, Bd. 61, H. 4, 1904, S. 570.
- K. BARTH. Zur Therapie der Aphonía spastica vermittels mechanischer Beeinflussung der Glottisbildung. *Arch. f. Laryngol. u. Rhinol.*, Bd. 16, H. 1, 1904, S. 80.
- PAUL FAREZ. Suggestion during Narcosis produced by some Halogenous Derivatives of Ethane and Methane. *Journ. Ment. Pathol.*, Vol. v., Nos. 2-3, 1904, p. 61.
- J. A. MACDOUGALL. On the Prophylactic Use of Morphia in Cases of Severe Cerebral Injury. *Lancet*, June 25, 1904, p. 1785.
- LUCIEN PICQUÉ et JULES DAGONET. Chirurgie des aliénés. Tome troisième. Masson et Cie, Paris, 1904, 8 fr.
- ESCHLE. Die krankhafte Willenschwäche und die Aufgaben der erziehblichen Therapie. Fischer, Berlin, 1904, M. 4.
- OLIVA. Una proposta riguardo ai Penitensiarîi. *Ann. di Freniat.*, Vol. xiv, f. 2, 1904, p. 156.
- W. J. HOWELL. Care of the Insane. *Am. Medicine*, June 4, 1904.
- ALEXANDER. Zur chirurgischen Behandlung der peripheren Facialis-Lähmung. Propfung des Facialisstammes an den Nervus hypoglossus. *Arch. f. Ohrenheilk.*, Bd. 64, H. 1 u. 2, 1904, S. 1.
- E. LUGARO. Una proposta di terapia chirurgica nella pazzia morale. *Riv. di Patolog. nerv. e ment.*, Vol. ix, f. 7, 1904, p. 306.

# **Review**

of

# **Neurology and Psychiatry**

---

## **Original Article**

### **NEUROMERES OF THE RHOMBENCEPHALON OF THE PIG.**

By O. CHARNOCK BRADLEY, M.B., F.R.S.E.,  
Royal Veterinary College, Edinburgh ; Goodsir Fellow, University of  
Edinburgh.

SINCE 1828, the year in which v. Baer (1) was the first to observe their occurrence, a considerable amount of attention has been directed to those segments of the embryonic neural tube for which Orr (11) suggested the name of "neuromeres." The earlier authors detected neuromeres in the region of the medulla oblongata only ; but it has been shown that they are not confined to this particular section of the central nervous system, but that the whole neural tube is divided into segments by constrictions which are indicated on the outer surface by grooves running transversely to the long axis of the tube. It was in 1889 that McClure (16) averred that segments could be distinguished in the embryonic spinal cord, and employed the name of "myelomeres" for these in order to differentiate them from the segments of the brain, which he named "encephalomeres." He was careful to point out that "myelomeres" and "encephalomeres" have common histological characters.

The earlier observers, moreover, attributed no particular significance to this segmentation ; indeed, Bischoff (2), in a figure illustrating the median section of a twenty-five days dog embryo,

showed seven folds in the wall of the medulla oblongata without making any reference to them in the text.

The following table shows in chronological order the animals in which neuromeres have been observed in the hind-brain, and the number of them which has been counted. Since the present communication is only intended to place on record the consideration of a small number of embryos of one particular mammal, it does not seem justifiable to preface it by a lengthy review of the somewhat numerous papers, both long and short, which have been devoted to the examination of neuromeric segments during the past half-century. Nor would such a proceeding be entirely necessary under any circumstances, for the literature has been very fully reviewed by Hill (28) in 1900, and still more recently by Kupffer (30).

	Year.	Investigator.	Animal.	No. of Neuromeres in Hind-Brain.
2 <sup>1</sup>	1842	Bischoff . .	Dog . .	7
3	1855	Remak . .	Chick . .	5-6
4	1869	Dursy . .	Calf . .	6
5	1875	Dohrn . .	Teleosts . .	8-9
6	1877	Mihalkovics .	{ Rabbit . . Chick . . }	5-6
7	1884	Béraneck . .	Lizard . .	5
8	1885	Rabl . .	Chick . .	7-8
			{ Teleosts . . Sheep . . Mouse . . Man . . }	5
9	1885	Kupffer . .		
10	1887	Béraneck . .	Chick . .	6
11	1887	Orr . .	Anolis . .	6
12	1887	Scott . .	Petromyzon .	5
13	1889	Prenant . .	Pig . .	6
14	1889	Platt . .	{ Chick . . Salmon . . }	6 5
15	1889	Hoffmann . .	{ Lizard . . Tropidonotus . }	7
16	1889	McClure . .	{ Chick . . Anolis . . }	6
17	1890	Reighard . .	Amblystoma .	5
			Stizostedion .	6

<sup>1</sup> The numbers in the first column refer to the list of papers given at the end of the present communication.

	Year.	Investigator.	Animal.	No. of Neuromeres in Hind-Brain.
18	1891	Zimmermann .	{ Rabbit . . . Chick . . . Acanthias . . . Mustelus . . . }	8
19	1891	Clarke . . .	Alligator . . .	5
20	1891	Waters . . .	{ Amblystoma . . . Gadus . . . }	5 6
21	1892	Froriep . . .	Mole . . .	7
22	1892	Herrick . . .	Snake . . .	6
23	1893	Kupffer . . .	Acipenser . . .	5
24	1895	Locy . . .	Squalus . . .	6 (9)
25	1895	Broman . . .	Man . . .	7
26	1898	Neal . . .	Squalus . . .	5
27	1899	Neumayer . . .	Sheep . . .	5
28	1900	Hill . . .	{ Chick . . . Teleosts . . . }	6
29	1903	Lewis . . .	Pig . . .	5

From an examination of the above table it is evident that the occurrence of neuromeres in mammalian embryos has not been extensively observed. I have only been able to find two references to neuromeres in the pig, and of these only one is at all a detailed account. Prenant (13) found six folds in the hind-brain of a 14 mm. pig embryo, and noticed that certain cranial nerves were connected with different folds. More recently, Lewis (29), in his description of a model of a 12 mm. pig embryo, says that "the hind-brain possesses three well-marked neuromeres followed posteriorly by a fourth shallow one. In a 9 mm. pig there are five which are distinct."

In view of the fact that little has been written on the neuromeres of the pig, it appears permissible to briefly relate some observations on the hind-brain of three embryos of different ages. It is unfortunate that the youngest of the three is already fairly advanced in development, it having been taken from the uterus nineteen days after coition. In this embryo the pontine flexure has barely begun to form. Looked at from the interior, the hind-brain shows seven grooves of unequal size, but symmetrically disposed. These grooves correspond to bulgings which are easily distinguished on the exterior of the neural tube.<sup>1</sup>

<sup>1</sup> These descriptions are based upon reconstruction models, made according to Born's method, as well as upon microscopic sections, both sagittal and coronal.

The first neuromere is large, and coincides in position with the, as yet, rudimentary Anlage of the cerebellum (Fig. 1). Its internal groove is not very deep, nor is it, in direction, perfectly transverse to the long axis of the hind-brain. Supposing the rhombencephalon to lie horizontally, the groove runs downwards and forwards. A very considerable external elevation corresponds to this internal groove. The groove and its correlated elevation lie at some distance from the fissura rhombesencephalica, which circumstance might lead one to question if the neuromere be really the first of the hind-brain. A cursory inspection might give rise to the supposition that a neuromere, previously evident anterior to the one now described, had lost its identity as a consequence of the incipient growth of the cerebellum. It has been shown, however, by Orr (11), Hill (28), and Kupffer (30), that the cerebellum is formed from one neuromere only (*Kleinhirneuromer* of Kupffer). It may, therefore, be concluded that the position and direction of the groove are due to growth of the cerebellum in its initial stages. Support is lent to this conclusion by the association of the various cranial nerves with the different neuromeres, as will be described later.

The internal groove of the second neuromere is, in this embryo, deeper than any of its fellows. The external constriction between the second and third neuromeral segments is absent, a condition possibly accounted for by the fact that the root of the N. trigeminus is connected mainly with the second neuromere, but also slightly with the third. The two neuromeres, consequently, are associated in the formation of one prominent external elevation, which corresponds in position to the widest part of the rhombencephalon. The fourth neuromere is marked by an internal groove of some depth, and a very prominent external ridge. The internal grooves and the corresponding external prominences of the fifth and sixth neuromeres are not so pronounced as are those immediately in front of them. They are, however, sufficiently distinct to prevent any dubiety as to the extent of these neuromeres.

The seventh segment of the hind-brain differs in some respects from its fellows. In the first place, it is larger than any of the others, with the exception of the first. It has a deep and spacious internal depression, which can hardly be called a groove, and its external eminence is equally extensive. Further-

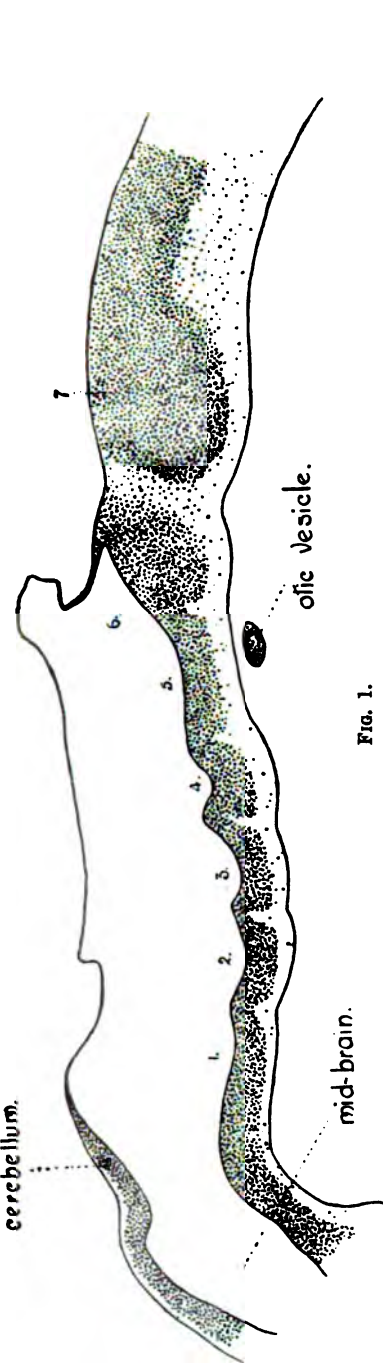


FIG. 1.

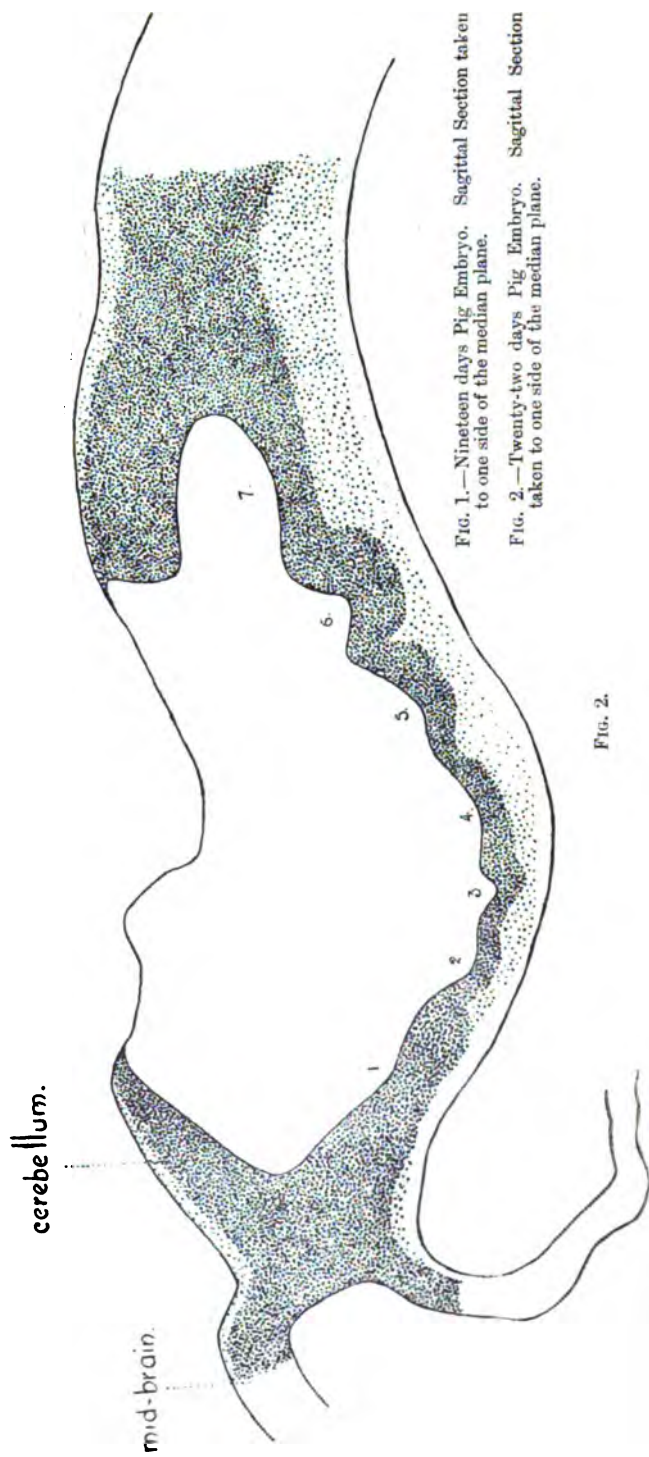


FIG. 2.

FIG. 1.—Nineteen days Pig Embryo. Sagittal Section taken to one side of the median plane.

FIG. 2.—Twenty-two days Pig Embryo. Sagittal Section taken to one side of the median plane.

more, the groove between the alar and basal laminae is continued forwards into the depression of the neuromere; but, while this is so, the posterior limit of the seventh segment can be determined by noting the greater depth and the more ample vertical dimensions of the neuromeral depression. It may be contended that this is not a hind-brain neuromere at all. Hill (28) has very firmly asserted that there are never more than six neuromeres in the rhombencephalon, and has averred that those who have counted more than six have included "segments caudad to the true sixth." It is, none the less, difficult to say definitely where the limit of the embryonic hind-brain is to be set. If the root-origin of the vagus is included in the hind-brain (Locy), the seventh neuromere, as here described, belongs to the rhombencephalon. If, however, the auditory invagination is taken as occurring on a level with the junction of the head and trunk (Neal and others), then the seventh neuromere does not belong to the brain. However the division of the head and trunk may be finally decided, in the embryo at present under consideration there is a clear cervical flexure which occurs behind the seventh neuromere. The relatively large size of the seventh segment might give origin to a further doubt. It is possible that more than one segment is here represented. This, clearly, could only be decided by the examination of younger material.

In a twenty-two days embryo (Fig. 2) there is no difficulty in again detecting seven neuromeres. The internal depressions are clear, but the constrictions and the intervening prominences on the exterior have become much less pronounced. It is, therefore, necessary to direct attention particularly to the internal grooves. The first groove is now relatively less deep, and, owing no doubt to the development of the pontine flexure, is more oblique in direction than it was in the previous embryo. The second groove is still the deepest, and this has doubtless been contributed to by an increase in height of the ridge between this groove and the first. In Fig. 2 no outstanding depth of the groove is indicated, but sections taken farther away from the median plane show it in a striking manner.

There is nothing remarkable about the third and fourth grooves, and the fifth and sixth, as was noticed in the nineteen days embryo, are, in the main, shallow as compared with the two immediately preceding them. Fig. 2 includes the deepest part



of the fifth and sixth grooves. The seventh depression is distinctly shallower, relatively, than in the nineteen days embryo, and the interzonal sulcus (sulcus between the alar and basal laminæ) is continuous with it. But, again (as in the nineteen days specimen), its greater vertical diameter serves to establish its identity. It should be added that the seventh neuromere is still decidedly larger than those immediately in front of it.

In a twenty-five days embryo all external evidence of neuromeres has disappeared, and only five grooves can be distinguished in the interior. There can be no doubt that those which have disappeared are the sixth and seventh. This can be determined by an examination of the connections of the cranial nerves. The interzonal sulcus can now be followed as far forwards as the fifth neuromeral groove; but, it is to be noted, it is much wider on a level with the sixth and seventh neuromeres than it is farther back.

The first groove is now very shallow, but occupies the same relative position as before. If this is indeed the remains of the internal groove of the first neuromere of the hind-brain, and there appears to be little reason to doubt it, then it is clear that this particular neuromere develops differently from those behind it. If we assume that the neuromeral groove lies originally equidistant from the two ends of the segment to which it belongs, then, in the second to seventh neuromeres inclusive, development has been equally vigorous in front of and behind the groove. In the cerebellar neuromere (first *Rautenhirn-neuromer* or *Kleinhirnneuromer* of Kupffer), on the other hand, growth has been particularly marked in that part which lies anterior to the groove. Consequently the groove has been gradually removed farther and farther from the fissura rhombomesencephalica. The groove of the second neuromere is now far and away the deepest, and the ridge between it and the first is more prominent than in the younger embryos.

It is not proposed at this time to follow the history of the neuromeral grooves further, but it may be stated that the examination of older embryos leaves little ground for doubting that some of them persist for a much longer time, in the pig at any rate, than is generally supposed.

The idea that the neuromeres are indicative of the primitive segmentation of the head has gained many adherents during the

past twenty years. The majority of those who have devoted special attention to these structures appear to be persuaded that they cannot be neglected in formulating a hypothesis as to the constitution of the vertebrate head. On the other hand, there are the utterances of those who have not been convinced of the segmental significance of neuromeres. Mihalkovics (6) looked upon them as being produced mechanically by the pressure of the mesoblast. Hertwig (31) made passing mention of them in the second edition of his text-book on embryology, and considered that their transitory nature was opposed to a conception of their segmental value. Gegenbaur (32) also did not consider neuromeres to be important morphological structures. Froriep appears, from his earlier papers, to have been inclined to attribute metameric value to them, but later (21) he has shown himself to be sceptical. In 1898 Neal (26) published his critical papers on the segmentation of the nervous system of *Squalus acanthias*, in which he stated that the neuromeres are not "segments in the true sense of the word," because, he says, "I find them irregular in size, inconstant in number, bilaterally asymmetrical, and without definite relation to structures known to be segmental."

In seeking to determine how much value should be assigned to neuromeres as affording evidence of the segmentation of the head, their association with cranial nerves has, naturally, received much attention. It would occupy an undue amount of space to detail the various differences in nerve-connection which are to be found in the reports of the different investigators. It will suffice, at this time, to make chief mention of the points upon which the majority are agreed, and incidentally touch upon the disagreements.

The N. acustico-facialis affords the most convenient starting-point, since all observers, with the exception of Locy and Broman, have described it as being associated with the fourth neuromere of the hind-brain. Broman (25) places it one neuromere farther back, *i.e.* in connection with the fifth; and Locy (24) states that the "roots of the facialis and the auditory arise separately in *Squalus acanthias*," the former from the fourth and the latter from the fifth hind-brain neuromere. In the embryos which form the subject of the present communication, the acustico-facialis root-complex is undoubtedly connected with the fourth neuromere.

The almost complete uniformity in the description of the origin of this particular nerve is of very considerable importance, since it thus forms a definite point from which the neuromeres can be counted.

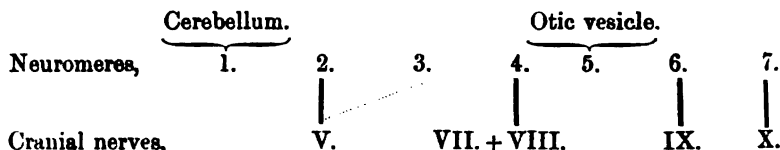
With the exception of Miss Platt and Broman, investigators are agreed that the primary connection of the N. trigeminus is with the second neuromere of the hind-brain. Miss Platt (14) holds that it arises from the constriction between the second and third neuromeres, and Broman (25) associates it with the third neuromere alone. Locy (24) and Hill (28) state that the anterior root of the trigeminus is related to the first neuromere, but the main root, they say, stands connected with the second. As has been previously stated, in the nineteen days pig embryo the N. trigeminus is mainly connected with the second neuromere, but in addition some of its fibres pass to the third. The relationship with the third neuromere is still more evident in the older embryos. It appears likely, therefore, that the third neuromeral connection is a secondary one, a similar arrangement having been noticed by Béraneck (10), Zimmermann (18), and Froriep (21).

It is generally conceded that the otic vesicle lies opposite the fifth neuromere, and in the nineteen days pig embryo this is certainly the case, though it also overlaps a small portion of the sixth neuromere. In the twenty-two days embryo the vesicle still lies opposite the whole of the fifth neuromere, but it now extends farther backwards than in the younger specimen, and so comes to cover a considerable part of the sixth neuromere. A backward movement of the otic vesicle during its development has been noticed by Locy (24) in *Squalus*, an observation supported by Kupffer (30).

The glosso-pharyngeus and vagus nerves were found in the pig embryos to be connected with the sixth and seventh neuromeres respectively. It should be stated that these nerves, as well as the acustico-facialis, have only very slender fibrillar connection with the neural tube in the nineteen days' embryo. In the next older specimen the connections are much stronger.

The precise origin of the N. abducens has offered difficulties to all investigators, it having been variously attributed to the third, fourth, and fifth neuromeres. The pig embryos do not afford any evidence of value.

The relations of the hind-brain neuromeres in the pig may be schematically indicated as follows :—



Prenant (13) is the only writer who has treated of the neuromeral nerve-connection in the pig; and, if we add the cerebellar neuromere to the six which he has described as occurring in a 14 mm. embryo, he is in almost entire agreement with what I have found and noted above. His only point of difference is in assigning an origin for one of the roots of the vagus to the sixth neuromere.

#### REFERENCES.

1. v. Baer, K. E. *Ueber Entwicklungsgeschichte der Thiere*, Th. 1, Königsberg, 1828.
2. Bischoff, T. L. W. *Entwicklungsgeschichte der Säugethiere und des Menschen*, Leipzig, 1842.
3. Remak, R. *Untersuchungen über die Entwicklung der Wirbelthiere*, Berlin, 1850-55.
4. Duray, E. *Zur Entwicklungsgeschichte des Kopfes des Menschen und der höheren Wirbelthiere*, Tübingen, 1869.
5. Dohrn, A. *Der Ursprung der Wirbelthiere und das Princip des Functionwechsels*, Leipzig, 1875.
6. v. Mihalkovics, V. *Entwicklungsgeschichte des Gehirns*, Leipzig, 1877.
7. Béraneck, E. "Recherches sur le développement des nerfs crâniens chez les Lézards," *Recueil Zool. Suisse*, T. 1, 1884.
8. Rabl, C. "Bemerkung über die Segmentirung des Hirnes," *Zool. Anz.*, Bd. 8, 1885.
9. Kupffer, C. "Primäre Metamerie des Neuralrohrs der Vertebraten," *Sitzb. d. Akad. München. Phys.-Math. Cl.*, 1885.
10. Béraneck, E. "Étude sur les replis médullaires du Poulet," *Recueil Zool. Suisse*, T. 4, 1887.
11. Orr, H. B. "Contribution to the Embryology of the Lizard," *Journ. Morph.*, vol. 1, 1887.
12. Scott, W. B. "Notes on the Development of Petromyzon," *Journ. Morph.*, vol. 1, 1887.
13. Prenant, A. "Note sur l'existence des replis médullaires chez l'embryon du porc," *Bull. de la Soc. d. Sc. de Nancy*, Sér. 2, T. 9, 1889.
14. Platt, J. B. "Studies on the Primitive Axial Segmentation of the Chick," *Bull. Mus. Comp. Zool. Harvard*, vol. 17, 1889.
15. Hoffman, C. K. "Ueber die Metamerie des Nachirns und Hinterhirns, und ihre Beziehung zu den segmentalen Kopfnerven bei Reptilienembryonen," *Zool. Anz.*, Bd. 12, 1889.
16. McClure, C. T. W. "The Primitive Segmentation of the Vertebrate Brain," *Zool. Anz.*, Bd. 12, 1889.
17. Reighard, J. "Development of the Wall-Eyed Pike, *Stizostedion vitreum*," *Bull. Michigan Fish Comm.*, 1890.

18. Zimmermann, W. "Ueber die Metamerie des Wirbelthier-Kopfes," *Verhandl. Anat. Gesellsch. (München)*, 5, 1891.
19. Clarke, S. F. "Habits and Embryology of the American Alligator," *Journ. Morph.*, vol. 5, 1891.
20. Waters, B. H. "Primitive Segmentation of the Vertebrate Brain," *Quart. Journ. Micros. Sci.*, vol. 33, 1892.
21. Froriep, A. "Zur Frage des sogenannten Neuromerie," *Verhandl. Anat. Gesellsch. (Wien)*, 6, 1892.
22. Herrick, C. L. "Embryological Notes on the Brain of the Snake," *Journ. Comp. Neurol.*, vol. 2, 1892.
23. Kupffer, C. "Die Entwicklung des Kopfes von *Acipenser sturio*, an Medianschnitten untersucht," *Studien zur vergleichenden Entwicklungsgeschichte des Kopfes der Kranioten*, Heft 1, München u. Leipzig, 1893.
24. Locy, W. A. "Metameric Segmentation in the Medullary Folds and Embryonic Rim," *Anat. Anz.*, Bd. 9, 1894.
- Ibid.* "Contribution to the Structure and Development of the Vertebrate Head," *Journ. Morph.*, vol. 11, 1895.
25. Broman, L. "Beschreibung eines menschlichen Embryos von beinahe 3 mm. Länge, mit speziellen Bemerkung über die bei demselben befindlichen Hirnfalten," *Morph. Arbeiten vom Schwalbe*, Bd. 5, 1895.
26. Neal, H. V. "The Segmentation of the Nervous System in *Squalus acanthias*," *Bull. Mus. Comp. Zool. Harvard*, vol. 31, 1898.
27. Neumayer, L. "Studie zur Entwicklungsgeschichte des Gehirnes der Säugethiere," *Festschr. z. 70. Geburtstag v. C. v. Kupffer*, 1899.
28. Hill, C. "Developmental History of the Primary Segments of the Vertebrate Head," *Zool. Jahrb. Abth. f. Anat. u. Ontogenie*, Bd. 13, 1900.
29. Lewis, F. T. "The Gross Anatomy of a 12 mm. Pig," *Amer. Journ. Anat.*, vol. 2, 1903.
30. v. Kupffer, C. "Die Morphogenie des Centralnervensystems," *Handbuch der vergleichenden und experimentellen Entwicklungslehre der Wirbelthiere*. Herausgegeben von Dr Oscar Hertwig. Lief 16, Jena, 1903.
31. Hertwig, O. *Lehrbuch der Entwicklungsgeschichte des Menschen und der Wirbelthiere*, 2te Aufl., Jena, 1888.
32. Gegenbaur, C. "Die Metamerie des Kopfes und die Wirbeltheorie des Kopfskelettes," *Morph. Jahrb.*, Bd. 13, 1887.

## Abstracts

### ANATOMY.

**THE ACTION OF PYRIDIN UPON THE NERVOUS TISSUES, AND (286) METHODS FOR THE SELECTIVE STAINING OF THE ENDO-CELLULAR FIBRILLAR RETICULUM AND PERIPHERAL RETICULUM OF THE NERVE CELLS OF VERTEBRATES.** ARTURO DONAGGIO, *Annali di Neurologia*, Anno xxii., f. 1-2, 1904.

IN this paper Donaggio describes a series of thionin methods for the demonstration of the neurofibrils and of the peripheral reticulum of the nerve cells. They are essentially elaborations of the methylene blue method described by him in 1896, which served to show the

presence of an internal and external reticulum, but was in certain respects unsatisfactory. He has since especially found advantage in the use of pyridin, both as a fixing and hardening agent and as a mordant. This advantageous action is manifested, as far as he has been able to ascertain, exclusively with thionin staining. He states in considerable detail the experimental data upon which his methods are based, describing especially the effect upon the staining result of pyridin alone and of this reagent followed or preceded by ammonium molybdate and sublimate.

He classifies his methods in five groups, according to the special tissue-elements of which they serve to give a selective coloration. He describes in all eight different methods. It must suffice to give here the details of the three processes which appear to be of chief importance, namely, those designated Methods III., IV., and VII. The first two of these result in selective staining of the endocellular fibrillar reticulum and of the long fibrils; the last serves for the demonstration of the peripheral reticulum.

The details of Method III., which is specially recommended for the nerve cells of the spinal cord, medulla, pons and spinal and sympathetic ganglia, are as follows:—

1. Fix and harden pieces about 5 mm. in thickness in pyridin for five or six days. Change the pyridin at least once.

2. Wash the pieces for 24 hours in several changes of distilled water. After some hours cut the pieces to a thickness of from 2 to 3 mm. The last washing should be carried out in a separate vessel, in order the better to insure the complete removal of pyridin.

3. Place the pieces for 24 hours in a 4 per cent. solution of ammonium molybdate in water, to which there has been added one drop of pure hydrochloric acid to each gramme of ammonium molybdate. The pieces should be placed on edge, and their position should be changed after 12 hours. The molybdate should be obtained, not in the powdered form, but in pieces. These must be dissolved in the necessary amount of water a considerable time before the solution is to be used. If the weather is warm, it is necessary to add the hydrochloric acid to a strong solution of molybdate (8 per cent. at least), and afterwards to reduce the strength to 4 per cent. It is also recommended to cool the molybdate solution before adding the acid.

4. Wash the pieces in water for 2 to 4 minutes; change the water once or twice. Then imbed in paraffin, thus:—Ordinary alcohol, 6 hours; absolute alcohol, 12 hours (change once); xylol, 8 to 10 hours (change once); xylol and paraffin, about 6 hours; paraffin melting at 45° for a longer or shorter time, according to the size of the pieces. If the weather is very warm, an additional immersion in paraffin melting at 50° may be necessary. A tem-

perature of 52° should not be exceeded. Cut sections of a thickness of from 3 to 7  $\mu$ . Reject the first few. Mount the succeeding ones on clean slides with the aid of distilled water. Dry the sections on the slide by means of slight heat. They should be placed on the top of a stove or incubator, and subjected to a temperature of from 35° to 40°. They must not be covered over in any way while drying. When the sections are dry, free them from any particles of dust that may have fallen upon them by means of a soft brush. Dissolve out the paraffin with xylol. Then wash the sections successively in absolute alcohol, ordinary alcohol, and water. The immersion in water should last for only 15 or 30 seconds. Next place the sections in solution of thionin in water of the strength of 1 in 10,000. This solution must be recently prepared, and care should be taken that the powder is entirely dissolved. The staining passes through the following three stages:—

- (1) Light bluish diffuse coloration of the grey and white matter;
- (2) Bright violet coloration of the grey matter;
- (3) Reddish violet coloration of the grey matter; pale bluish coloration of the white matter.

These stages are passed through in from 3 to 30 minutes, according to the temperature. The staining having reached the third stage, the succeeding procedure may be one or other of two:—

- (a) Pass the sections successively through distilled water, ordinary alcohol, absolute alcohol, and xylol, and then mount in Grüber's neutral balsam.
- (b) Distilled water, ordinary alcohol, distilled water (the alcohol must be thoroughly washed out), the ammonium molybdate solution for 15 to 30 minutes, distilled water for 15 to 20 minutes (renew the water two or three times), ordinary alcohol, absolute alcohol, xylol, neutral balsam, coverglass.

The former procedure yields preparations showing a sharp colour contrast between reddish violet fibrils and a light blue ground substance. The latter, which the author prefers, gives a sharper staining of the fibril, but the colour contrast is wanting.

Method IV. is recommended specially for the cerebrum and cerebellum, but may be used for other nervous tissues with good results. The details differ from those of Method III. in the following respects:—The fresh tissues are fixed in a pyridin and pyridin nitrate mixture. This is made by adding slowly to 72 parts of pyridin, 28 parts of a 50 per cent. aqueous solution of nitric acid. Heat is evolved as a result of the chemical combination, and the fluid must be allowed to cool before it is used. The tissues remain in this solution for 24 hours, and are then transferred to pure pyridin, in which they lie for 36 hours. The subsequent treatment differs very slightly from that in Method III. For the cerebral

cortex it is preferable to fix the sections upon the slide without the aid of heat. For the cerebellum it is recommended that some should be fixed to the slide in this way, and others with the aid of heat. For both tissues the immersion in water, after removal of the paraffin, should be shorter than in Method III., as should also the stay in the various fluids subsequent to staining in thionin solution.

Method VII., for the staining of the pericellular reticulum, is applicable to the whole of the cerebro-spinal axis. The directions are as follows:—

1. Place pieces of tissue, from 2 to 3 mm. in thickness, in Heidenhain's corrosive sublimate solution for 24 hours. Remove excess of sublimate by washing the pieces for 24 hours in saturated solution of iodine in water. Then wash in distilled water for 2 to 3 hours.

2. Place in pyridin for 36 to 48 hours. Change the pyridin at least once.

3. Distilled water for 24 hours. Change several times. Ammonium molybdate solution for 24 hours.

4. Distilled water for half to one hour. Pyridin again for 36 to 48 hours (change once).

5. Place the pieces in thionin solution of the strength of 1 in 10,000, or 1 in 15,000, for 48 hours. The pieces must be suspended in the solution. This may be done by attaching a piece of cork to one edge of the block with the aid of melted paraffin. Renew the colouring solution after 24 hours.

6. Suspend the pieces, still attached to the corks, in ammonium molybdate solution for 24 hours.

7. Wash in distilled water, renewed several times, for 24 hours. Pass through alcohols and xylol, and imbed in paraffin. Cut sections from 5 to 15  $\mu$  in thickness. Remove paraffin with xylol and mount the sections in neutral balsam.

Donaggio concludes from the results obtained with these methods that there are at least two types of nerve cells, namely, those provided with only an endo-cellular reticulum, and those, forming by far the larger proportion, which have two systems of conduction, consisting of fibres forming the endo-cellular reticulum and of fibres traversing the cell and maintaining their individuality. The methods hitherto employed have not given a complete demonstration of these two structural arrangements. Bethe's method reveals only the long fibrils, and the conclusions which have been founded on the results of this staining process are to a large extent invalidated by the demonstration of the existence of an endo-cellular reticulum. Donaggio hesitates to form any definite conclusion regarding the difficult questions of the anastomosis of the long fibrils and the communication of these fibrils with the internal



reticulum. He refers to the results recently obtained with Cajal's new silver method, and gives reasons for doubting that this process serves to reveal the internal reticulum satisfactorily. He also alludes to some experimental observations, carried out along with Fragnito, which show that in certain secondary lesions, whilst with Cajal's method the neurofibrils appear to be completely destroyed, in preparations by his own methods they are still visible though altered as regards their affinities for the thionin stain.

As regards the pericellular reticulum, his more recent observations confirm the view he has previously expressed, namely, that it is of neuroglial nature, although certain ray-like structures lying in the meshes of the reticulum are probably nerve-endings.

W. FORD ROBERTSON.

**RAMON Y CAJAL'S NEW FIBRIL METHOD.** M. v. LENHOSSEK,  
(287) *Neurolog. Centralbl.*, Nr. 13, 1904, p. 593.

THIS is mainly a critical examination of the two opposing theories regarding the minute structure and relations of the nerve cells in the light of the results recently obtained by Cajal. It is contended that the new evidence virtually confirms the theory of simple contact, and correspondingly discredits that of continuity of fibrils from cell to cell. The allegation that the fibrils are continued beyond the points to which they can be traced in Golgi- and Bethe-preparations, is one that has never been established by observation, and it seems inconsistent with the appearances presented by the fibrils in the new silver preparations. The evidence of the various methods taken together, in the author's opinion, justifies the conclusion that the fibrils really end where they appear to end. By the application of his new method to the nervous system of the leech, Cajal has been able to show an arrangement of the neurofibrils differing from that described by Apáthy in this invertebrate. What has been alleged to be an elementary network, appears in silver preparations to be simply a plexus of fibrils, showing no more evidence of anastomosis than is to be found in Golgi-preparations. The speculations about the "nervous grey" and the "Golgi-networks," Lenhossék dismisses as not belonging to the region of positive histological observations. Lastly, the fact that the method can be applied successfully to the nervous system of young embryos, has rendered it possible to obtain proof that the neurofibrils are formed at a very early period. At an early stage of development they are less numerous, but individually more distinct, than at a later period, and form a comparatively wide-meshed reticulum. The author has himself observed them in a human foetus of 25 mm. This evidence of the early existence

of the neurofibrils proves that the hypothesis, according to which they are not developed in the nerve cells, but from special fibril-forming cells, is quite untenable. W. FORD ROBERTSON.

## PHYSIOLOGY.

**THE NEURONE THEORY.** G. DURANTE, *Rev. Neurol.*, June 30, 1904, (288) p. 573.

ANOTHER paper by this author with the same title has already been abstracted in this journal. In it he proposed that as the neurone theory is no longer tenable and is in disagreement with more recently ascertained facts, the nervous system should be regarded as composed of functional units, for which he proposes the name *neurules*, polycellular in origin, and analogous to the lobules of a glandular structure.

The present paper is in part a reiteration in part a completion of his earlier arguments. It is best to abstract it under the several headings into which he has divided it:—

1. *Continuity or discontinuity of the terminal fibrils.*—Apáthy, Bethe, and Nissl have described continuity. Cajal, on the other hand, supported by Déjerine, Azoulay, and Marinesco, has, from preparations by his new method, pronounced against continuity, but this is not conclusive, as
  - (a) his method is only partial; as all impregnation methods it is not identical with but only complementary to true staining. The impregnation is probably limited to the interfibrillar protoplasm, and from pictures so obtained we cannot conclude on the termination of the fibrils.
  - (b) On the polycellular conception of the neurule the continuous axiscylinder results from the fusion of differentiated portions of several neuroblasts. Should this fusion be in places incomplete, it does not invalidate the polycellular theory.
2. *Segmental and autogenous regeneration.*—The progressive elongation of the axiscylinder of the central stump of a sectioned nerve, which has been described by the upholders of the neurone theory, cannot be followed in preparations. Further, it is not in agreement with the biological law that the new growth of a cell only takes place from its less differentiated protoplasm. On the other hand, discontinuous segmental regeneration has been frequently demonstrated. This peripheral regeneration takes place from the inter-nodal protoplasm round the sheath of Schwann and slowly proceeds from the periphery toward the point of interruption, the nervous impulses necessary for its complete

differentiation coming from the periphery probably through the "net" of Bethe and Apáthy.

3. *Recurrent sensibility and nervous substitution.*—

(a) Physiological experiments have demonstrated that the peripheral end of a sectioned anterior root is sensible and that this sensibility takes place through the posterior roots. This recurrence of the sensation does not occur through the point of junction of the anterior and posterior roots, as it is lost after section of the combined nerve, so it must be peripheral. This is unintelligible if there be free and terminal ending of the nerves. As the sensation is always referred to the distribution of the sensory portion of the nerve, it cannot be due to local stimulation of the sensory supply of the motor root.

(b) The several well-authenticated cases in which no sensory loss resulted from lesion of a large nerve trunk containing sensory fibres, and in others the rapid narrowing of the anæsthesia following such a lesion, make it necessary to assume, if the neurone theory be held, that the neighbouring intact nerves contain sufficient fibres for the supply of the distribution of the injured nerve. These facts, however, could be more easily explained by the assumption of the existence of a peripheral nervous system analogous to vascular capillaries, through which there could be anastomosis of the peripheral ends of the various nerve trunks.

4. *Propagation of degeneration.*—Monakow, Mœbius, and others have drawn attention to the alteration of one neurone as indirect result of the disease of another. Schäffer and Marinesco have also demonstrated chromatolysis in the anterior cornual cells in recent hemiplegia. Bræunig (and surely Warrington?) have found changes in the same cells after section of the dorsal roots.

Thus the limitation of degeneration to the one neurone seems to be the exception rather than the rule, and cannot be used as an argument for the neurone theory.

5. *Conclusions.*—The arguments involved in support of the neurone theory are no proof of the cytological unity of the neurone, but only stand in favour of it being a physiological unit. The conception of a nervous system composed of primitive polycellular masses analogous to the lobules of glandular organs is best in agreement with known facts. While each cell of such a lobule (*i.e.* the constituent neuroblasts) has an individual life, and reacts individually to pathogenic agents, it is also dependent on anything which affects the lobule (neurule) as a whole.

It is proposed to drop the term neurone, which is an anatomical

term based on the misinterpretation of facts, and to supplant it by the term *neurule*, by which to imply the physiological unity of the nervous lobule.

GORDON HOLMES.

**THE INNERVATION OF THE SPHINCTERS AND MUSCULATURE**  
(289) **OF THE STOMACH.** W. PAGE MAY, *Journ. of Physiol.*, Vol. xxxi., Nos. 3 and 4, June 1904, p. 260.

THE author carried out a research at University College, London, on a little over thirty anæsthetised cats, dogs, and monkeys. The chief method he employed was the so-called graphic method which had been used by Bayliss and Starling in their investigation of the intestinal movements. He passed one, or usually two small india-rubber tambours up into the stomach through a small aperture in the duodenum. These tambours could be distended at will to any required size, and lay in contact with the inner lining of the stomach; hence they naturally followed all the movements of the stomach wall, and recorded by means of the ordinary kymograph, not merely the condition of the muscular tonus of the stomach wall, but also all gastric movements, and at the same time gave accurately the exact time relations. The author found that on stimulating the peripheral end of a divided vagus nerve, the gastric movements at once ceased and the muscular tonus diminished, and that this was followed by a strong gastric contraction, or series of contractions, with an increased tone of the gastric musculature. The inhibitory period lasted sometimes during the whole of the period of stimulation, sometimes for a period of only 10 to 15 seconds, and was then followed by the augmentor effect. The left vagus exerted a greater influence than the right vagus, both as regards inhibitory and motor effects. The arterial pressure was always recorded during the period of experiment, and the results obtained by the author agree in the main with those of previous observers, but amplify them in many details.

As a result of similar investigation of the influence of the splanchnic nerve on the stomach, the author obtained divergent results from nearly all previous workers; many of whom, such as Schiff, Morat, Openchowski, and others, obtained either a motor effect, or an inhibitory effect, or both effects. The author found, as a result of his experiments, that neither the splanchnic nerves, the sympathetic chain in the thorax, nor the rami communicantes running to the stellate ganglion showed any direct influence whatever, either motor or inhibitory, on the gastric movements.

Similarly he stimulated at one time or another all over the cerebral cortex in anæsthetised monkeys and dogs, but could not obtain by the apparatus and method used any evidence of alteration of the gastric tone or movements.

He also investigated the innervation of the cardiac and pyloric sphincters of the stomach by means of a column of physiological saline solution, the height of which as maintained by the cardiac sphincter could be easily recorded. In other experiments the passage of each drop of saline solution through the pyloric sphincter was automatically recorded by means of a tambour. In this way he showed that the effect of vagus and splanchnic stimulation on the cardiac and pyloric sphincters is practically the same as on the stomach.

AUTHOR'S ABSTRACT.

## **PATHOLOGY.**

**A CONTRIBUTION TO THE PATHOLOGY AND PATHOGENESIS (290) OF CHOKED DISC.** By Dr KAMPHERSTEIN, Weimar (from the University Eye Clinic, Breslau), *Klin. Monatsbl. f. Augenheilk.*, June 1904, p. 501.

AFTER touching briefly on the various theories which have been propounded to explain the production of choked disc, Dr Kampherstein gives an account of the microscopical examination of 55 eyes derived from 44 cases, in which the condition had been observed *intra vitam*. Most of the eyes were obtained from the collection of Professor Uthoff. Fifty-one of the eyes were derived from cases of cerebral disease: tumour cerebri 44 eyes, cerebral syphilis 1, cysticercus cerebri 2, actinomycosis 1, solitary tubercle 1. In one case there had been chronic nephritis and apoplexia cerebri crescens. Of the 4 remaining eyes 3 were cases of orbital tumour and 1 a case of papillitis following necrosis of the cornea from diphtheria.

Dilatation of the sheath space round the optic nerve was present in 65 per cent. of the cases of brain disease. In 35 to 40 per cent this distension was very marked. The widening of the space affected usually the subarachnoid portion, the arachnoid being thickened and pressed against the dura. In 3 eyes a subdural space could be identified, and in 1 of these the sheath space appeared divided into two by the arachnoid. Usually the dilatation was greatest just behind the eye. In longitudinal sections it was noted that sometimes the dilatation was present only on one side of the nerve.

In 38 out of 51 cerebral cases inflammatory changes were found in the nerve sheath. In very early cases these changes were usually absent. The round cell infiltration when present was often in patches, sometimes diffuse. In the three cases of choked disc from orbital tumour the appearances did not differ materially from those found accompanying brain tumours.

In 5 eyes out of a total of 50 derived from cases of cerebral tumour the optic nerves were found quite free from inflammatory changes, although 4 of these showed dilatation of the nerve sheath, and 1 a perineuritic infiltration.

Inflammatory changes in the optic nerve itself were noted in 56 per cent. of the eyes examined. In many of these, however, the changes appear to have been slight. Dr Kampherstein considers the oedema the most characteristic feature of choked disc. It was present to a greater or less extent in 60 per cent. of the eyes examined. The type of oedema most commonly noted he describes as interfascicular, involving the connective tissue of the septa, and being often very marked round the central blood-vessels. An intrafascicular oedema he describes as much rarer, and only existing along with the previous type. Dr Kampherstein strongly maintains the view that these evidences of oedema revealed by the microscope are real and not merely due to hardening processes, section cutting, etc. He never found similar evidence of oedema in sections of normal optic nerves. The atrophy following choked disc he describes as a neuritic degeneration. The connective tissue of the septa, infiltrated with leucocytes in the acuter stages, subsequently becomes thickened. A slow disappearance of the nerve fibres is accompanied by an increase of neuroglial elements. In fresh cases the vessels of the nerve are normal. Later their walls become thickened.

Particular attention was paid to the position of the lamina cribrosa. In 88 per cent. of fresh cases Dr Kampherstein found it driven forwards. The part of the lamina which shows this "Vorbuckelung" best is the anterior or choroidal portion.

Of 42 choked discs examined microscopically, 15 showed no inflammatory changes. In the others round cell exudation was noticed, commencing round the blood-vessels in early cases. In more advanced and severe cases the infiltration of the connective tissue with round cells was very marked, especially in the region of the lamina cribrosa. The physiological excavation was observed in many cases to be filled up by a fine connective tissue. The oedema of the disc extended about a disc's breadth into the surrounding retina, causing it to appear swollen and folded on section.

Dr Kampherstein does not discuss the theories of optic neuritis at any length. He thinks that pressure of lymph in the sheath space cannot alone explain choked disc as dilatation of this space was present only in 40 per cent. of his cases. Against the inflammatory theory he cites the occurrence of optic neuritis in chronic hydrocephalus and in some published cases of hæmorrhage into the nerve sheath. He supposes that choked disc is due to an

oedema propagated along the nerve from the brain. A sort of incarceration of the nerve head by the rigid scleral ring aids doubtless in increasing the swelling of the disc.

A valuable series of literary references is appended to the article.  
J. V. PATERSON.

### CLINICAL NEUROLOGY.

**ON DIABETIC NEURITIS.** F. W. PAVY, *Lancet*, July 2nd, 1904, (291) p. 17, and July 9th, 1904.

THE writer gives a brief history of our knowledge of this condition, which is of quite recent date. This type of neuritis corresponds generally with neuritis due to other causes, the most prominent and earliest noticed symptom being impairment of the knee-jerks, but it presents peculiar features of its own.

The symptoms include motor, sensory, reflex, vaso-motor and nutritional phenomena. Among the motor symptoms are cramp (which also comes on in the course of diabetes independently of neuritis, apparently as a direct result of the sugar circulating in the tissues), loss of co-ordination, producing staggering gait and ataxia, which, however, is not so marked as that of tabes, fibrillary tremors, and squint due to neuritis of the oculo-motor nerves. Sensory symptoms include hyperæsthesia, numbness, tingling, lightning pains, girdle sensation, and various other paræsthesiæ, while anginal pains, which the writer has met with in diabetes, he attributes to neuritis of the pneumogastric nerves. The most important reflex symptom is the loss or impairment of the knee-jerks, which may reappear as the diabetes becomes amenable to treatment; the Argyll-Robertson pupil and visceral derangements found in tabes do not form part of diabetic neuritis and so serve to differentiate the conditions. Among the vaso-motor symptoms, localised sweatings are common in diabetic neuritis, while the trophic disturbances include perforating ulcers and gangrene of the toes and other parts, especially liable to follow slight injuries when neuritis is present, and allied, in the writer's opinion, to Raynaud's disease. These latter symptoms must be carefully distinguished from other nutritional symptoms due to direct action of the sugar upon the affected tissues, *e.g.* cataract, xanthoma, boils.

The writer fully discusses the agency by which the neuritis is produced. He quotes Ross and Bury to show that the general view is that some unknown "toxic agent," other than sugar, constitutes the cause, but he speaks emphatically from his own experience, which goes to prove that the occurrence of neuritis and the presence of a large quantity of sugar go hand in hand, neuritis appearing rather in those people who suffer for a long time

from the "alimentary" type of the disease than in very acute cases in young persons. He attributes much the same place to sugar as alcohol holds in the alcoholic variety of multiple neuritis.

As to treatment, he finds the condition very amenable in general to reduction of sugar in the system by diet, and opium, and he attaches importance to the use of iodide of potassium and bromide of ammonium. For the relief of pain he recommends the galvanic current and liniment of aconite. JOHN D. COMRIE.

**NEURITIS PERIAXILIS.** BERNHARDT, Berlin, Sonderabdruck aus (292) der *Deutsch. Ztschrift. f. Nervenheilk.*, Bd. xxvi.

THIS paper makes no claim to add anything of importance to our knowledge of the subject it deals with, but rather to review and correlate the successive observations which have led to its recognition. The facts which it discusses concern the finer changes that occur in the peripheral nerves in certain types of neuritis, and it deals with them more specially from the clinical aspect. They may be summarised as follows: In a considerable number of cases that can be classed under the term neuritis the axis cylinder of the nerve may persist whilst the myelin sheath perishes, a condition being thus established which differs in many important respects from ordinary Wallerian degeneration. The latter may occur in certain nerves, whilst others in the same patient show the less serious change, or the milder type alone may be present.

It has been studied in poisoning by lead, mercury, and alcohol, and also in diphtheria and sepsis, as well as in some paralytic conditions of childhood, and in the spinal neuritic type of progressive muscular atrophy. The point of chief clinical interest is that where the nerves are thus affected muscles which respond freely to volition are irresponsible to electrical stimulation, or exhibit the reaction of degeneration. This can be explained on Erb's hypothesis that voluntary impulses pass along the axis cylinder, but electrical stimuli are conducted by the myelin sheath.

The paper commences with a reference to Erb's case, published in 1875, and the first section is occupied with a review of the clinical observations that have accumulated since that date. These include several by Prof. Bernhardt himself, amongst which are some instructive cases of traumatic neuritis of the median nerve, where marked electrical changes were accompanied by but slight impairment of voluntary movement.

The second section of the paper deals with the occurrence of a similar type of failure in response to electrical stimuli in healthy young animals and infants at the period prior to full development of the myelin sheath, to which attention was directed by Soltmann as early as 1878; the third section discusses the results obtained



from experimental work on animals, and especially refers to the researches of Gombault, who, almost a quarter of a century ago, as the outcome of both experimental and clinical observations, published conclusions which are practically identical with those recorded in this paper. The last section reviews the facts observed by various pathologists in post-mortem examinations of cases of progressive muscular atrophy.

The paper will be of service to those who wish to trace the history of this interesting subject, although its usefulness is impaired by its prolixity and involved style. At the close there is a literature index which is fairly satisfactory up to 1899, but is deficient in more recent references.

H. RAINY.

**PARALYSIS OF THE OCULOMOTOR NERVES IN A CASE OF (293) TABES DORSALIS.** A. PAPADAKI, *Rev. Neurol.*, June 30, 1904, p. 585.

**AFTER** referring to the frequency of ocular affections in tabes, either partial or complete, temporary or permanent, the author draws attention to the little definite knowledge we possess of their causation.

He describes the case of a tabetic general paralytic, who, eighteen months before death, suddenly developed paralysis of the left oculomotor nerve, which soon again disappeared, only inequality of the pupils persisting. There was a recurrence of the ptosis and squint twenty-one days before his death which did not disappear.

In the autopsy he found basal meningitis, especially dense and thick round the left oculomotor nerve, and marked arteriosclerosis of the basal vessels. On microscopical examination the third pair of nuclei seemed to be normal (Nissl's method not employed), and the trunks of both nerves were stained well with Weigert-Pal method.

On the left side, however (that of the palsy), there were several small hæmorrhages in the trunk of the nerve, and marked dilatation of the small vessels in and around it. The walls of the larger vessels round the extra-medullary portion of the nerve had undergone much degeneration and in parts such aneurysmal dilatation as must have compressed the nerve, and the meninges too were thickened and infiltrated. The intraorbital part of the nerve was not examined.

The author comes to the conclusion that the clinical symptoms of this case may have been due to the simultaneous action of the three agents, *i.e.* the hæmorrhage into the trunk of the nerve, basal meningitis, and compression by arteriosclerotic vessels, but he regards the former as the more immediate cause and so explains the sudden onsets.

GORDON HOLMES.

**THE DIAGNOSIS AND TREATMENT OF TABES.** CONSTER, *Berlin*.  
(294) *klin. Wochenschr.*, July 4 and 11, 1904, p. 782.

SPECIAL attention is directed to the early diagnosis of the earliest symptoms of tabes, and the results of 93 cases are taken to support the belief that in the earliest stages thorough anti-syphilitic treatment is not without value.

S. A. KINNIER WILSON.

**A CASE OF "SUBACUTE COMBINED SCLEROSIS," WITH  
(295) PROFOUND ANÆMIA.** JAMES TAYLOR, *Brain*, Spring 1904,  
p. 27.

THE case here published is that of a woman æt. 63, seen first in July 1903 on account of difficulty and unsteadiness in walking. It had been noticed by her friends that she had become much more pale and sallow in the last few years. She had been conscious of weakness since December 1902. She was ataxic, had active knee-jerks, and no sensory impairment, but subjective sensations of tingling and numbness. She became rapidly worse, and was admitted to hospital on September 14, 1903. She then had active knee-jerks, ankle clonus on each side, and the plantar response was extensor. She was unable to stand, and there was sensory impairment over the lower limbs and a distinct girdle sensation. On September 23 the lower limbs became flaccid and the knee-jerks were abolished. The sphincters became paralysed, the sensory impairment became more distinct, and extended as high as the second rib. She wasted rapidly, she wandered at night, and she finally died of exhaustion on September 27.

Apart from spinal cord changes, the organs were normal. The changes in the cord are figured in the article, and consist of extensive sclerotic changes in the anterior, posterior and lateral columns in the cervical and dorsal regions. In the lumbar region the sclerosis is practically confined to the lateral columns.

The writer refers to various papers published since Dr Bowman's and his own in 1895, the starting-point of the work done on this subject being Lichtheim's and Minnich's researches between 1887 and 1893, and Putnam's paper in 1891. Although Lichtheim's researches dealt with cases of pernicious anæmia, there seems no doubt that although such cord changes are found in some cases of what is regarded as pernicious anæmia, other conditions of anæmia and malnutrition are also found with so-called "subacute combined sclerosis," the name given to the condition by Russell, Batten, and Collier. Reference is made to the work done by these observers, and also by Mott, Putnam and Taylor in America, and by Adami, Pitren, Marburg, Göbel, Dana, Bastianelli, and others.

The opinion is expressed that the disease is one almost as definite in its symptoms and pathology as tabes, and like the sclerotic conditions found by Williamson in diabetes, and by Tucek and others in pellagra, probably the result of a toxic blood condition. The writer, however, thinks that the sclerosis of the cord produced by the toxic condition may be aggravated in parts of the spinal cord by the occurrence of small hæmorrhagic foci, similar to those occurring, *e.g.* in the retinæ in cases of profound anæmia or of toxic blood states. He discusses the question whether the cases of ataxic paraplegia described by Gowers belong to this group; and although Gowers' cases have a longer clinical history than many cases of subacute combined sclerosis, he is inclined to the view that they do, as do also the cases described by Putnam in 1891 of "system sclerosis of the spinal cord, with diffuse collateral degeneration occurring in enfeebled persons past middle life, and especially in women." In several of the latter the clinical course is considerably longer than *e.g.* in the cases published by the writer, in this respect more closely resembling Gowers' cases of ataxic paraplegia. The changes in the spinal cord are probably the same in all, although the clinical course may vary considerably both in duration and in symptoms.

AUTHOR'S ABSTRACT.

**MULTIPLE SCLEROSIS.** SPILLER and CAMP, *Amer. Journ. Nerv. (296) and Ment. Dis.*, July 1904, p. 433.

AFTER referring to the infrequency of the disease in America as compared with Europe, the authors give an account of two cases which came under their care. Case No. 1 appears to have been of the myelitis type, there being sensory, motor, and reflex signs affecting the lower limbs; but no disorder of speech and no nystagmus; vision in the right eye, however, appeared to be diminished. Whilst in hospital the woman threw herself out of a third story window, sustaining a fracture-dislocation of the spine which caused her death. Post-mortem: the right optic nerve appeared almost completely degenerated, and sclerotic patches were found throughout the spinal cord. The microscopical examination was, however, complicated by recent degeneration due to the fracture.

Case No. 2 was of the ataxic-paraplegic type. Sensation was normal everywhere, sight was poor, and slight nystagmus was present. Subsequently speech became slow, but not scanning; and intention tremors appeared in the upper limbs, with impairment of tactile sensation in the lower limbs and trunk. Microscopical examination revealed sclerotic areas throughout the cerebro-spinal axis.

The writers remark upon the frequency with which the optic tract is implicated in this disease; and there is no doubt that a careful ophthalmoscopic examination often leads to a correct diagnosis of disseminated sclerosis being made in cases where the symptoms at first appear to be purely spinal, and it must be remembered that pallor or even atrophy of the discs may exist even when the vision is not complained of.

With regard to the pathology, the authors consider it extremely difficult to determine the relation of multiple sclerosis to multiple myelitis, although they apparently consider the indefinite limitations of the patches in their second case to be evidence of the identity of these two pathological conditions. Undoubtedly this is so, and in a recent paper contributed to this Review by the writer of this abstract, it was shown that the definite or indefinite circumscription of the patches is largely a question of the activity of the morbid process and age of the respective patches. Both ill and well defined patches may occur in one case, but the morbid process invariably appears to start in, and to be essentially a degeneration of, the myelin substance. A. F. TREDGOLD.

**LEPROSY OR SYRINGOMYELIA!** DELUPIS, *Wien. med. Wochenschr.*, (297) June 4, 1904.

A CASE at first diagnosed as leprosy (for no apparent adequate reason) is shown to be syringomyelia as evidenced by scoliosis, hemianæsthesia, and other sensory disturbances, absence of exanthems, absence of thickened nerves, absence of Hansen's bacillus in the blood, skin, and nasal mucus.

S. A. KINNIER WILSON.

**A YEAR OF LUMBAR PUNCTURES IN A GENERAL HOSPITAL.** (298) CHAUFFARD and BODIN, *Gaz. des Hôpitaux*, June 28, 1904, p. 725.

A RESUMÉ of this nature is of real value because it treats alike of failure and success, and deals avowedly with the clinical aspect of the question. 223 lumbar punctures were made: in very exceptional cases did headache ensue as a sequela; in three cases there was vomiting. No further complication of any sort occurred. Much of the results is confirmatory of previous work, e.g. as far as tabes, general paralysis, tubercular meningitis, zona, syphilis, etc., are concerned. In 14 cases the liquid on being withdrawn was more or less tinged with blood, and an attempt is made

at a differential diagnosis between a pure meningeal hæmorrhage and a cerebro-meningeal hæmorrhage.

Considerable stress is laid on the value of lumbar puncture in cases of ear mischief (*cf.* also *Review of Neurology and Psychiatry*, April 1904, p. 334). Lymphocytes in the fluid in cases of earache probably indicate a mild meningitis, if associated with headache, bradycardia, etc. Instances are given of the curative value of lumbar puncture in zona, cerebro-spinal meningitis, tubercular meningitis, labyrinthine vertigo, etc.

S. A. KINNIER WILSON.

**CYTOTOLOGICAL EXAMINATION OF THE CEREBRO-SPINAL (299) FLUID.** SCHLESINGER, *Deutsche med. Wochenschr.*, July 7, 1904, p. 1022.

A RESUMÉ of the examination of the cerebro-spinal fluid in 38 cases of nervous disease. Nothing is added to what is already well known.

S. A. KINNIER WILSON.

**ON DONATH'S METHOD OF DEMONSTRATING THE PRESENCE (300) OF CHOLINE IN THE CEREBRO-SPINAL FLUID OF EPILEPTICS.** MANSFELD, *Hoppe Seyler's Zeitschr. f. physiol. Chemie*, Bd. 42, 1904, S. 157.

MANSFELD criticises Donath's method of obtaining choline (*cf.* *Review of Neurology and Psychiatry*, Jan. 1904, p. 62). He holds that the identity of Donath's crystals with those of choline is doubtful. He obtained similar forms (of which photographs are given) in the urine of the dog and in the normal cerebro-spinal fluid of the horse, and believes that they are due to the combination of platinum chloride (used in the test) with an ammonium salt. No reference is made to the work of Swale Vincent and Cramer (*Journal of Physiology*, Nov. 1903).

It must not be forgotten, on the other hand, that choline is certainly present, though in very small quantities, in normal cerebrospinal fluid, and further, Gumprecht has shown that it is probably excreted by the urine.

S. A. KINNIER WILSON.

**CHOLINE IN THE CEREBRO-SPINAL FLUID AS A SIGN OF (301) NERVOUS TISSUE DEGENERATION.** KINNIER WILSON, *Rev. Neurol.*, April 30, 1904, p. 401.

ATTENTION is drawn to the clinical value of the test for choline as elaborated by Mott (for the blood) and Donath (for the cerebro-

spinal fluid). Details of the method are given, with its results in thirty cases of nervous disease, in tabes, cerebral hæmorrhage, Jacksonian epilepsy, hysterical paraplegia, transverse myelitis, etc., etc. Its value as corroborative of organic disease is emphasised. A full cytological examination was made of each case, but no relation between this and the choline content of the fluid was detected.

AUTHOR'S ABSTRACT.

**PHOSPHORIC ACID IN THE CEREBRO-SPINAL FLUID IN**  
 (302) **GENERAL AND IN NERVOUS DISEASES.** DONATH,  
*Hoppe Seyler's Zeitschr. f. physiol. Chemie*, Bd. 42, 1904,  
 S. 141.

JAKSCH in his well-known handbook states that '01 to '02 per cent  $P_2O_5$  is normally present in the cerebro-spinal fluid. The writer using Neumann's method for the determination of phosphoric acid found '17 per cent. in a case of tumor cerebri, '2 per cent. in tabes and as much as '5 per cent. in tabo-paralysis. Apparently one may acknowledge a connection between more or less active nervous tissue destruction and the amount of  $P_2O_5$  in the cerebro-spinal fluid. Reference is made to the work of Widal, Sicard, and Ravaut (*Revue Neurologique*, 1903), on the presence of a globulin in normal cerebro-spinal fluid, with the addition of a serum albumen in tabes and general paralysis. An analogy is drawn between the two sets of facts.

S. A. KINNIER WILSON.

**POST-DIPHTHERITIC CHRONIC BULBAR PARALYSIS.** WILFRID  
 (303) HARRIS, *Lancet*, July 23, 1904, p. 209.

AFTER referring to two similar cases which he published in *Brain*, winter number 1903, the author describes a third case which has come under his notice. The patient was shown at a meeting of the Medical Society of London in April last, and resembles a case published by Stadthagen in 1884.

A man, æt. 25, who had, seven years before being seen by Dr Harris, complained of "relaxed throat," which he said followed on scarlet fever. He had noticed the throat trouble return six weeks after the rash, when it became extremely painful, and six weeks later difficulty in swallowing, with nasal regurgitation, commenced. Since then patient has never been able to swallow dry food, and has had much difficulty even with his saliva. He had been slightly worse for the past six months, and has had slight diplopia twice, but food does not now pass down the nose. There is marked

paralysis of the orbicularis oris, the tongue is slightly atrophied and cannot be fully protruded. The soft palate does not move on phonating A, although it rises on reflex stimulation and on phonating O. Glottis was normal. No other face muscles were affected. There was slightly reduced faradic irritability in the orbicularis oris, but nothing further, and there was no myasthenic reaction. Large doses of strychnine were administered, and galvanism applied locally to the pharynx and lips, and in six weeks considerable improvement resulted.

The author thinks that this case is due to a slight nuclear lesion, and is similar to the other two cases published in *Brain*. He argues that the absence of ptosis, weakness of jaw and other muscles, excludes myasthenia gravis. There is some doubt as to the diphtheritic history in all three cases, and the author remarks that septic sore throats due to streptococcic invasion have been known to give rise to paralysis. Of the three cases two have recovered, one completely after a six years' history, and this one partially after seven years. The third case, the most severe of the group, is slowly getting worse, the palatal palsy being complete, the laryngeal commencing, while eyelids and lips remain completely paralysed.

ROBERT A. FLEMING.

**URÆMIC HEMIPLEGIA, WITH CHANGES IN THE NERVE  
(304) CELLS OF THE BRAIN AND CORD, etc. T. H. WEISEN-  
BURG, *Amer. Journ. Nerv. and Ment. Dis.*, July 1904, p. 456.**

In this paper the writer gives an account of the microscopical examination of the central nervous system in two cases of "uræmic hemiplegia." In Case 1, the patient, 71 years of age, died fourteen days after the onset of left hemiplegia. Post-mortem: there were found chronic nephritis, cardiac hypertrophy and dilatation, and arterio-sclerosis. The chief changes in the nerve cells consisted of swelling and chromatolysis of the cells of Purkinje, and intense pigmentation of Betz' cells; there was also chromatolysis of some of the latter, most marked on the right side. In the cord the cells of Clarke's column were much degenerated, and those of the anterior horns much pigmented; there was also Marchi degeneration of the left crossed pyramidal tract traceable upwards as far as the internal capsule.

In Case 2, the patient, 77 years of age, died four days after the onset of left hemiplegia, there being some spasticity of the right leg also. Post-mortem: granular kidneys, fibroid myocarditis, chronic endarteritis and atheroma of the cerebral basal vessels were found. Under the microscope, there was chromatolysis of some of the Betz' cells of the *left* side, many, however, being nearly normal,

also slight changes in Purkinje's cells. The cells of the anterior horns were about normal, and there was no tract degeneration.

The author contends that these changes in the nerve cells were the result of uræmic intoxication; but to the writer of this abstract such a view hardly seems to be warranted by the facts. Indeed, quite apart from the unsuitability of cases of this age and presenting these pathological lesions for the investigation of such a point, the existence of uræmia in these two cases is not made absolutely clear, and in the absence of any record of convulsions, or any mental state characteristic of uræmia, it is certainly unfortunate that in each case the notes of the condition of the urine should have been lost.

Although it is highly probable that in acute uræmia there may be toxic bodies capable of injuriously affecting the neurones (to which the observations of Acquisto and Pusateri, Donetti, and Ewing, quoted by the author, lend support), the changes in these two cases can hardly be due to such an acute condition, since they are for the most part of a chronic nature. And whilst it is not improbable that a latent and chronic uræmia may bring about changes like those described, it is to be remembered that the same may result from long-standing vascular disease. Considering that in each of these cases arterio-sclerosis was present, undoubtedly accompanied in one by atheroma of the cerebral vessels, the conclusion that the nerve changes are characteristic of the uræmic condition hardly seems to be justified. A. F. TREDGOLD.

**A CASE OF "ATYPICAL" TETANY COMMENCING WITH CON-  
(305) TRACTION OF FACIAL MUSCLES. S. H. SCHEIBER,  
*Wien. med. Wchnschr.*, Juni 25 u. Juli 2, 1904, S. 1206 u.  
1270.**

SCHEIBER first refers to a case of tetany occurring in a nursing woman, previously described by him (*Wien. med. Wchnschr.* Nov. 5, 1903), which showed certain abnormal features; and he then gives details of another case of "Atypical" tetany in a seven months' pregnant female, æt 34, in which the illness first manifested itself in the face, and not, as is usually the case, in the extremities, contractions in the latter only supervening six months later. The patient was in her seventh pregnancy, but had never had a living child (two abortions and four still-born), the husband being probably syphilitic. The cramps began in the face in May 1902, not during, but two months previous to the onset of pregnancy, and they remained continuously and exclusively confined to the facial and occipito-frontalis muscles until November, by which time tonic contractions supervened in the upper and lower extremities,



and also in the intercostals. Another peculiarity of the case was the fact that the cramps were continuous (tetania continua) rather than paroxysmal (one to two weeks without intermission), but were absent during sleep. The phenomena in the face and hands were not of equal intensity on the two sides, but were symmetrical in the case of the trunk and lower extremities. The symptoms of Trousseau, Erb, and Chvostek were obtained, and the author notes the presence of a reflex contraction of the lower lip on the same side, elicited on stroking the side of the face, and also produced by percussing any of the facial muscles of the same side, no contraction, however, being got in the muscle struck ("paradoxical contraction").

As long as the tetany remained confined to the face, the patient's general health did not suffer, but when the disease spread to the extremities, she became very ill (headache, sleeplessness, loss of appetite, etc.).

With energetic antisyphilitic treatment the patient improved, and gave birth to a full-time living child.

W. E. CARNEGIE DICKSON.

**CONTRIBUTION TO OUR KNOWLEDGE OF TETANY IN DILATATION OF THE STOMACH.** RICHARTZ, *Ztschr. f. Klin. Med.*, Bd. 53, 1904, S. 369.

CASE.—Female, æt. 32. Usual clinical picture of gastric dilatation, e.g. feeling of load and delayed pain after meals, vomiting, loss of weight and strength, etc. No albuminuria. An attack of tetany supervened after vomiting, the neck, back, hands (typical "accoucheur" position), and lower limbs, but not the face, being affected. These tetanoid seizures were repeated, and during one of them a hard, irregular, nodular resistance could be felt in the neighbourhood of the pylorus. Trousseau's symptom present; Chvostek's phenomenon not elicited. Test meal after attack contained lactic, but no free hydrochloric acid. Laparotomy was subsequently performed, and when the stomach was washed out on the morning before the operation, two small fragments of plum-stone were brought up. On opening the abdomen a tumour (colloid cancer) about the size of a small hen's egg was found at the pylorus, and in the dilated stomach a mass containing no fewer than eighty small plum-stones, fragments of several others, and a cherry-stone was found, the extraction of which took so long that only a gastro-enterostomy could thereafter be performed. These foreign bodies must have lain in the patient's stomach for at least fifteen months, perhaps longer. Richartz then goes on to dis-

cuss the causation of the disease, and refers to the various theories that have been suggested, *i.e.* :—

1. Fleiner's reflex theory.
2. Chronic auto-intoxication from stagnating stomach contents, acting on the nervous system, *cf.* ergot, lead, etc.
3. Küssmaul's theory of inspissation of the blood and abnormal dryness of the tissues.

After the operation, the patient had no more tetanoid seizures, probably owing to prevention of the stagnation, and therefore of the possibility of further production of toxic substances in the stomach. One important fact left unexplained by these theories is the rarity of gastric tetany as compared with the great frequency of dilatation of the stomach. It is possible that locality may play some part in the causation of the disease, perhaps owing to the presence of some infective condition.

W. E. CARNEGIE DICKSON.

**CONTRIBUTION TO THE SYMPTOMATOLOGY OF GASTRIC  
(307) CANCER AND THE OCCURRENCE OF TETANY IN  
DISEASES OF THE STOMACH.** HONIGMANN, *Ztschr. f.  
Klin. Med.*, Bd. 53, 1904, S. 347.

HONIGMANN here gives a very full account of a case of cancer of the pylorus in a man of 42, in which typical tetanoid phenomena supervened during the patient's stay in hospital. The attack commenced with vomiting, pains all over the body, stiffness of the arms, feeling of tightness of the skin, etc., followed by the characteristic "accoucheur" contraction of the hands, and involvement of the lower extremities, the attack lasting half-an-hour. Trousseau's and Chvostek's symptoms well marked. Pupils dilated. Trace of albumin in urine. The muscles of mastication were also involved in a subsequent attack, and the patient complained of loss of the power of vision, the pupils being now widely dilated, and reacting but feebly to light. Peristaltic contractions could be elicited on tapping over the stomach. Five days after the onset of tetany, the operation of pylorotomy and anterior gastro-enterostomy was performed, the patient leaving hospital nineteen days later. He died about four months after the operation, and on post-mortem examination colloid cancer of the pylorus (causing moderate stenosis), and secondary growths in the peritoneum, mesentery, omentum, and in numerous lymphatic glands, were found. There was also obliteration of the pelvis of the right kidney and upper part of the ureter, with atrophy of the right, and enlargement of the left kidney and commencing nephritis.

The author then discusses the case at great length, making

special reference to the presence of hyperacidity and hypersecretion found previous to, but not after, an attack of hæmatemesis which occurred two days before the onset of tetany. He also reviews the theories which have at various times been put forward to explain the disease.

W. E. CARNEGIE DICKSON.

**THE THYROID GLAND AND TETANY.** JOSEF JACOBI, *Wien.* (308) *klin. Wchnschr.*, Juli 7, 1904, S. 768.

THE author discusses the relationship of the function of the thyroid gland to myxœdema and exophthalmic goitre, and then goes on to consider its influence on the occurrence of tetany. Nathan Weiss was the first to record the occurrence of typical and severe tetanoid symptoms following extirpation of the gland, and later observations by subsequent writers have confirmed the relationship existing between the thyroid gland and tetany. Valsall and Generali assert that the loss of *thyroid* function leads to myxœdema, loss of *parathyroid* secretion to tetany. Other authors have recorded cases of tetany occurring in combination with myxœdema, with exophthalmic goitre, or with both of these diseases together. Jacobi then gives particulars of four cases of tetany occurring in patients with enlarged thyroids.

CASE I. Boy, æt. 16. Compositor's apprentice, complained of contractions in hands and feet, etc., for eight months. Thyroid soft and enlarged to size of hen's egg. Vertebral column showed a kypho-scoliosis. Electrical excitability of the muscles and nerves involved much increased. Symptoms of Chvostek, Trousseau, and Erb all present.

CASE II. Girl, æt. 15, sister of the above patient. Both lobes of thyroid enlarged to size of pigeon's egg; soft; enlargement began at age of five. Typical tetanoid attacks in both hands. Electrical phenomena, etc., as in Case I.

CASE III. Mother of Cases I. and II. Thyroid enlarged to size of hen's egg. Eyelids subject to frequent cramp-like contractions. Chvostek's and Erb's phenomena present. Trousseau's symptom absent. Slight paræsthesia of right arm. No tetanoid contraction. Severe pains all over body.

CASE IV. Female, æt. 32; married at 21; seven living, full-time children. Thyroid began to enlarge at third pregnancy. Many of the symptoms of exophthalmic goitre, with, in addition, the phenomena of Erb and Chvostek. The hands showed typical tetanoid contractions.

Jacobi finally refers to the various possible factors in the causation of the tetany in these cases, *e.g.* heredity, infection, lead poisoning, condition of thyroid, especially during pregnancy, etc.

W. E. CARNEGIE DICKSON.

**THE OCCURRENCE OF TETANY ALONG WITH MYOTONIC**  
 (309) **SYMPTOMS.** V. VOSS, *Deutsche Ztsch. f. Nervenheilk.*, Bd. 26,  
 H. 4-6, 1904, S. 521.

CASE I. Boy æt. 17. Had suffered for last three years from stiffness in muscles of legs; difficulty in going upstairs, etc. This became more marked a year ago, and the upper limbs also became affected, e.g. on grasping anything with the hand he had difficulty in letting go again. Paræsthesia (formication) present. Muscles of neck also affected. No enlargement of stomach or of thyroid gland. Usual symptoms of tetany also present (Chvostek, Hoffman, Trousseau). Tetanoid contractions observed in all the extremities. Muscular power normal. *Tache cérébrale* very marked. Electrical excitability of muscles and nerves increased and altered as in myotonia (Erb's myotonic reaction).

CASE II. Boy, æt. 18. For the last six months had suffered from stiffness of the muscles in walking, specially noticeable on starting the act; difficulty in going upstairs. Stiffness of tongue and muscles of mastication for a month, and difficulty in letting go objects grasped with the hands. No spontaneous tetanoid contractions. Symptoms aggravated by cold. Chvostek's symptom very active; Trousseau's phenomenon also present. No pathological changes found in any of the internal organs. Myotatic irritability increased. Electric excitability for Galvanic and Faradic current increased in the extremities. No myotonic reaction. At the first examination symptoms of both tetany and myotonia (Thomsen's disease) were found combined; later, Trousseau's and Chvostek's phenomena, and finally the myotonic condition also, disappeared.

W. E. CARNEGIE DICKSON.

**A CASE OF VERBAL BLINDNESS WITH AGRAPHIA. AUTOPSY.**  
 (310) DÉJÉRINE et THOMAS, *Rev. Neurol.*, July 15, 1904, p. 645.

DÉJÉRINE has distinguished two clinical varieties of verbal blindness, that associated with agraphia and due to lesion of the angular gyrus, and the form in which spontaneous writing and writing from dictation is preserved and the lesion only interrupts the fibres connecting the common visual centre with the angular gyrus.

The authors describe the case of a woman who, eight years previous to her death, suddenly developed right hemiplegia and hemianæsthesia, right hemianopia and became simultaneously unable to read. There was some temporary paraphasia, but her power of speech was soon quite good again. She understood all

said to her but was unable to read a word ; at the most could only name some letters. She was, however, able to write quite well, but was quite unable to read what she had written.

Two years later there was a second slight attack of apoplexy affecting the same side. After this her spontaneous speech remained quite good, she understood quite well, and could repeat words correctly, but she was absolutely unable to read or to write spontaneously or from dictation. She could not copy print into writing or calculate, but was still able to spell words aloud when asked to.

The patient finally died from a large hæmorrhage into the right hemisphere.

In the left hemisphere there was a large focus of softening, chiefly in the ventro-medial surface of the temporo-occipital lobe. On the external surface it was seen to involve the third temporal convolution and the posterior ends of the first and third occipital, while ventromesially it destroyed the cuneus, the fusiform, lingual and subcallosal gyri, the hippocampus and cornu Ammonis. The pulvinar thalami and internal geniculate body were also destroyed, but the angular gyrus remained intact.

From a series of microscopical sections which were made through the whole hemisphere, it was seen that the lesion included the area of distribution of the posterior cerebral and posterior thalamic arteries, and extended down to the ependyma of the posterior horn of the lateral ventricle. Involved in it were the thalamic radiations to the occipital lobe, to the posterior part of the parietal, and a considerable proportion of those to the temporal lobe, the inferior longitudinal bundle, the tapetum, the forceps minor and the callosal radiations which turn round the posterior horn of the lateral ventricle. The post-lenticular portion of the internal capsule, the pulvinar and other portions of the thalamus were also destroyed. There was some retrograde atrophy of the fillet, but the pyramidal tracts seemed normal. The chief points discussed are: (1) the existence of severe hemiplegia persisting eight years, which was undoubtedly organic (Babinski's sign was not then known), without direct involvement of the pyramidal system; (2) that agraphia (loss of spontaneous writing, of writing to dictation, and of the ability to copy) should exist without involvement of the angular gyrus or of any of the cortical centres of speech. The agraphia which results from lesions of the latter differs from that found in the present case by the inability of the patient to spell. In this case the patient could no longer write, not because she did not possess the memory of the normal arrangement of letters in words, but because she was unable to evoke the visual image corresponding to each letter, or the memory of the movement corresponding to the visual image of each letter.

As the patient had not lost the visual verbal images, it seems necessary to assume that there was an interruption between the centre of these (in the angular gyrus) and of that for the movements of the arm. As the right hemiplegia was so complete that she could only attempt to write with the left hand, the interruption must have been of the tracts connecting the left angular gyrus with the right Rolandic region.

It will be remembered that the agraphia only became added to the other symptoms after the second apoplexy, so presumably the lesion was at first limited to the occipital lobe and only later extended deeper and destroyed the commissural or projection fibres of the angular gyrus.

Both the clinical and anatomical descriptions are very complete and the latter is copiously illustrated by drawings.

GORDON HOLMES.

#### CONJUGATE DEVIATION OF THE EYES AND HEMIANOPIA.

(311) J. GRASSET, *Rev. Neurol.*, July 15, 1904, p. 645.

THE author opens his paper by the short account of the case of a man of sixty-two years of age who suddenly developed left hemiplegia, hemianæsthesia, and hemianopia, with constant deviation of the eyes to the right side and inability to move them to the left beyond the middle line. On the first day the head was also deviated to the right, but was turned to the left during the last six days of life. The patient only lived ten days after the onset of the illness. At the autopsy a hæmorrhage was found occupying the right optic thalamus and the neighbouring portion of the internal capsule.

His aim is to discuss the various standpoints taken in regard to the causation of conjugate deviation of the eyes in hemiplegia, especially with reference to Bard's view.

Grasset himself has maintained that this conjugate deviation is always the expression of a direct affection of the function of the apparatus which normally acts in turning the eyes to the one side, the result differing as the lesion is irritative or destructive; that inability to turn the eyes to the one side can only depend on direct motor palsy, while persistent conjugate deviation to the one side with destructive lesions is due to a loss of tone in the one set of muscles and relative excess in the opposite.

According to Bard, on the other hand (with Roux, Déjerine, and Marie), the conjugate deviation of the eyes in hemiplegia is of sensorial origin and is most frequently associated with hemiplegia. It is the automatic effect of unilateral central sensorial loss, the deviation being directly an active process of the sensori-motor centres of the healthy hemisphere. External impressions are not

necessary to this relative overaction ; it may rise from the spontaneous evolution of sensorial images. Bard regards the deviation in hemiplegia as different from that met with in pontine lesions, in tonic or clonic spasms originating in the epileptogenous zone of the motor cortex, and to post-hemiplegic contractures. Grasset's objections to this view are :—

1. It cannot explain the palsy of voluntary conjugate movement to the paralysed side, as was present in his case.
2. The paralytic conjugate deviation of the eyes to the one side, which occasionally follows convulsive deviation to the opposite side, is not intelligible with this theory ; but, accepting Grasset's theory, it is quite analogous to the other motor palsies which follow convulsion.

Bard has no justification for regarding the various forms of conjugate deviation of different nature.

3. A cortical centre for deviation of the head and eyes to the opposite side, which Bard denies, is well known to exist.
4. The loss of the tonus of the paretic muscles, which must exist to explain persistent conjugate deviation in hemiplegia, is not, as Bard asserts, peculiar to peripheral palsies, but is frequently present in those of central origin.

In Grasset's case the head and eyes were at first deviated in the same direction, but during the last six days of life the head was turned to the opposite side. This the author explains as due to the close anatomical association of the central tracts for the two movements, the lesion of that for the eye movements being destructive, and of that for rotation of the head irritative.

GORDON HOLMES.

**CEREBELLAR HEMIATAXIA AND ITS ORIGIN.** LUDWIG MANN,  
(312) *Monatsschrift f. Psychiat. u. Neurolog.*, June 1904, p. 409.

AN interesting case of hemiataxia of the left side, with no trace of sensory disturbances. Associated on the left side were Babinski's phenomenon and a fair degree of muscle wasting. Optic neuritis was markedly present, preventing the determination of the presence of a hemianopsia. Post-mortem a tumour was found limited to the left occipital lobe, and pressing on the cerebellum. A careful critical analysis is made of the case, with numerous references to experimental work on the cerebellum, demonstrating the association of unilateral cerebellar lesions with hemiataxia on the same side. In view of the clinical features of the case, it is unfortunate that the spinal cord was not examined.

S. A. KINNIE WILSON.

**THE DELIRIUM ALOOHOLICUM FEBRILE OF MAGNAN.**

(313) ALZHEIMER (of Munich), *Centralbl. f. Nervenl. u. Psych.*, July 15, 1904, p. 437.

VARIOUS authors give different statistics as to the frequency of the existence of fever in cases of delirium tremens; it is certain that in some cases there is no rise in temperature, in some other cases the rise can be explained by complicating circumstances. Magnan described a particular form of alcoholic delirium with marked rise of temperature; the existence of this form has been denied by Rose. Alzheimer gives briefly the notes of three cases out of the Frankfort clinic, which corresponded to Magnan's type. The temperature began practically with the onset of the delirium, in all cases an epileptiform seizure being an early symptom; the patients died 5, 8, 14 hours respectively after the onset of delirium, their temperatures being finally 103, 104, and 107. Microscopic examination in the two cases where a sectio was allowed, disclosed no extra-cerebral cause for the fever; the cortex of the brain was much damaged. The cases were clinically undoubted cases of alcoholic delirium with exceedingly rapid course. Alzheimer compares the fever in these cases to that in the status epilepticus, in the fits of general paralysis, and in brain-syphilis, where there is demonstrable a large destruction of brain tissue.

But although the clinical type of Magnan is true to the facts, such cases are merely the extremes of an uninterrupted series, at the other end of which is delirium tremens with no fever at all. The existence of such cases show that delirium tremens does not necessarily end in cure or Korsakoff's syndrome, but may in itself, without complications, cause the death of the patient.

C. MACFIE CAMPBELL

**PSYCHIATRY.****THE BASIS OF A CLASSIFICATION OF MENTAL DISEASES.**

(314) RONCORONI, *Ann. di Freniatria*, vol. xiv. f. 1, 1904.

WE have here a discussion of the question of the classification of mental diseases. In the present state of our knowledge of these diseases a complete and satisfactory classification is admitted by the author to be well-nigh impossible. He, however, approaches the subject from a broader point of view, and suggests a classification which fuller knowledge may amplify and complete.

He first criticises the criteria on which previous classifications have been based, and shows that they are insufficient for a rational scheme.

He deals with them in the following order—

A. Etiological criterion.—This is unsatisfactory, because no



cause produces characteristic mental forms, but these vary according to the soil that they act on, and according to the persistence and gravity of that action. Moreover, no mental disease is produced by a single cause. He points also to the various forms of mental disease which are produced by alcohol and other poisons, by old age, and by puberty, etc.

*B. Anatomopathological criterion.*—This is insufficient, because lesions of diverse nature, but affecting like structures, can give rise to identical morbid pictures, and identical lesions, which affect different structures, produce the most diverse syndromes. Moreover, our anatomicopathological knowledge is very imperfect, and it is impossible in this connection to neglect chemical and physicochemical conditions.

*C. A purely symptomatological criterion* is incomplete, because no single symptom is pathognomonic of a special mental disease.

*D.* He also shows that the biological, prognostic, and other criteria, which have been employed, are equally unsatisfactory.

We must remember that the nervous system is not a collection of cellular groups of equal value and function. The function may be of a higher or lower grade, and with this variation of function, there are also chemical and morphological differences, and also differences of resistive power.

It follows, therefore, that the more diffuse and more grave is the action of the morbid cause, and the longer it acts, the wider will the lesion spread, the more will it invade the less evolved centres, the more will the action of these centres tend to become automatic, and the more tendency will there be for the morbid state to become systematised and chronic.

The criteria then, which will indicate the greater gravity, will be:—

1. Precocity—index of gravity of morbid cause and less resistant soil.

2. The appearance not only of transitory phenomena, but of stable and systematised phenomena, without much tendency to recovery—index of recoverability.

3. The fact of its having invaded structures and functions less evolved—index of the profundity of the lesion.

4. Its having produced not only phenomena of excitement, but also of paralysis—nature of the functional lesion.

The term gravity may then be considered to include most of the criteria which have served as bases for the other classifications.

The author then refers to the two great categories of mental disease suggested by Morel—"parafrenie" (deviations of development or psychic degenerations) and "frenopatie." These can be largely distinguished by their respective gravity, and each form may be more or less grave. In the "parafrenie" the symptoms

are more systematised, and there is very rarely recovery without defect, although paroxysmal attacks may be overcome.

It is not, of course, to be understood that all "frenopatie" are recoverable. In the "parafrenie" also heredity plays a more important part.

Moreover, in the conditions of "arrest of mental development," to which the author applies the term "frenastenie," the manifestations are largely dependent on another factor of "gravity," viz., precocity. The distinguishing features of the "parafrenie," besides the heredity, are the following:—the unstable, eccentric, character, seen even before the mental disease declares itself, so that this represents only an accentuation of a previous condition—the slight gravity of the determining cause—the frequent appearance of exacerbations—the not rare presence of degenerative morphological characters—the frequent preservation of the coherence of ideas, and the small chance of recovery.

Then in the "frenasthenie" we may find not only a lesion of associative processes and of memory, but the perception of stimuli is altered, and also many stimuli may fail to be transformed into sensations and perceptions.

Recognising amongst the acquired diseases the two great categories of Morel, "parafrenia" and "frenopatia," and taking as an index of the profundity of the lesion, the want of or presence of primitive or automatic lesions of motion, the author further divides the "parafrenie" into psychic and nervous degenerations, and the "frenopatie" into psychoneuroses and cerebropathies.

By the term "primitive lesions of motion," it is to be understood that they appear in the first period of the disease, and by the term "automatic lesions of motion," it is to be understood that they are independent of the will.

Now the irregular movements of the maniacal and catatonic states are still to a certain extent dependent on the will, although not free. But epileptic seizures, unequal pupils, disarthria, and dysgraphia, fibrillary tremors and alternation of the knee jerks are automatic, i.e. independent of the will. These automatic movements are again divided into those of excitement (contractures, nystagmus, convulsions), and those of paralysis (changes of pupil, fibrillary tremors, etc.).

A last category of mental diseases includes some intermediate forms, and those forms connected with disease of some other organs.

It is true that very different pathogenic processes can produce lesions, which by their localisation and gravity are practically identical, and the author admits that this is a defect of his scheme. This may be remedied, however, by deeper knowledge.

But, according to this scheme, there exists a fundamental unity

of pathogenesis of the various mental diseases, and the distinction of the special forms lies in close relation to their gravity—using this word in its broadest sense—*i.e.* to the precocity, profundity, recoverability, and nature of an identical mechanism of action.

For purposes of diagnosis, then, we must take count of the morbid form, *e.g.* dementia, of the variety, *e.g.* catatonic, and of the cause, *e.g.* precocity, and the terms catatonic and precocious imply not morbid forms, but complements of the term dementia, conditions, that is, which underlie certain symptoms.

The author goes on to discuss many special morbid conditions, but this discussion cannot be reproduced here.

The following is the classification suggested by the author, in the form of a table:—

**A. ACQUIRED MENTAL DISEASES.**

“Parafrenie”

(deviations of development).

“Frenopatie.

(a) Without primitive or automatic lesions of motion.

“Parafrenie,” properly so-called.  
(Psychic degenerations.)

principally { periodic insanity.  
emotional { alternating insanity.  
                  circular insanity.  
                  affective insanity.  
                  moral insanity.  
                  rudimentary paranoia.

principally { paranoia and its varie-  
ideational { ties.

Psychoneuroses.

partial sensory excite- } “frenosi  
ment . . . } sensoria.”  
total ideo-motor excite- } mania.  
ment . . . } melancholia.  
ideo-motor depression . } amentia (confu-  
temporary general func- } sional insanity).  
tional ideo-motor arrest } dementia.  
permanent general ideo-  
motor arrest . . . }

(b) With primitive or automatic lesions of motion.

Neuroses (paroxysmal motor phenom-  
ena, chiefly of excitement).

phenomena less system- } hysteria.  
atised . . . }  
phenomena more sys- } epilepsy.  
tematised . . . }

Cerebropathies (motor phenomena, chiefly  
of deficiency).

acute . . . { delirium tremens.  
                  acute delirium.  
periodic or paroxys- } periodic cerebropathy.  
mal . . . }  
subacute or chronic { cerebroplegic dementia.  
                              progressive paralysis.  
                              pseudoparalysis.  
                              cerebral syphilis.  
                              cerebropathies, with  
                              macroscopic lesions.

**B. “FRENASTENIE” ARREST OF MENTAL DEVELOPMENT, CONGENITAL,  
OR IN THE FIRST YEARS OF LIFE.**

imbecility.  
idiocy.  
cretinism.  
microcephaly.

C. (a) Forms of passage between the various forms of mental disease.  
(b) Forms mixed with nervous diseases or diseases of other organs.

R. G. ROWS.

**PREDISPOSITION IN THE ETIOLOGY OF MENTAL DISEASE**

(315) E. M. DE MONTYEL, *Jour. de Neur.*, July 5, 20, 1904, p. 241.

THE author maintains that without a certain predisposition the various etiological factors in mental disease do not produce insanity. The predisposition may be congenital or acquired: a congenital predisposition is due either to heredity or to the condition of the parents during coitus, or of the mother during pregnancy. Under heredity the author traces the influence of various diseases in causing the hereditary predisposition to insanity; the three main hereditary origins are consanguinity of parents, tuberculosis, and the neuroses. Tuberculosis in parents is especially traceable in cases of melancholia and paranoia.

The predisposition to insanity may be acquired owing to typhoid fever and malaria, chronic alcoholism and lead poisoning, traumatism and insolation. The delirium of fever in individuals with no predisposition is not identical with that of individuals already predisposed; in the former the trouble is purely of the intellect, emotional and sensory disturbances are absent; while in the latter hallucinations play a large part, and the delirium takes on a peculiar dream-like character.

The predisposition, whether acquired or hereditary, is insufficient of itself to create a psychosis without an immediate cause, but it often gives a special character to the individual, who is more or less a degenerate. Previous authors have held that degeneracy, whether acquired or of hereditary origin, is characterised by want of mental equilibrium as well as some mental deficiency. The author makes a difference between acquired and inherited degeneracy: in the latter case there is a complete absence of intellectual enfeeblement and rather an exaltation of certain mental faculties: in this category he includes men of genius as superior degenerates. As the character of the predisposed varied with the origin of the predisposition, so the psychoses which arise from it vary according as the taint has been acquired or inherited.

C. MACFIE CAMPBELL

**QUANTITATIVE AND QUALITATIVE LEUCOCYTE COUNTS IN**

(316) **VARIOUS FORMS OF MENTAL DISEASE.** LEWIS C. BRUCE  
and A. M. S. PEEBLES, *Journ. Ment. Sc.*, July 1904, p. 409.

THE authors examined the blood of 150 patients for long periods at a time. In pure cases of *acute melancholia* no leucocytosis was observed, and a guarded prognosis should be given in cases of this affection where a hyperleucocytosis is observed early in the disease.

In *excited melancholia* a high leucocytosis occurs early in the disease. A relapse is generally preceded by a fall of the leucocytosis to 10,000 or 13,000, with a low polymorphonuclear percentage. As the excitement increases the leucocytosis and the percentage of polymorphonuclear cells rises until the attack reaches its height. On recovery the leucocytosis remains high, but the polymorphonuclear percentage generally falls to 60 or below 60.

The leucocytosis of *acute continuous mania* and *recurrent mania* in adolescent cases exactly resembles that of excited melancholia, and in all three diseases a persistent leucocytosis occurs upon recovery taking place.

During the depressed stage of "*folie circulaire*" a high leucocytosis with a polymorphonuclear percentage between 60 and 70 was observed. If a period of sanity followed the depression the leucocytosis remained high, but the polymorphonuclear percentage fell to 60. When excitement set in the leucocytosis fell at first, rose later as the excitement increased, and then gradually fell to normal. These observations point to the fact that the depression and excitement of "*folie circulaire*" are quite different from ordinary attacks of mania and melancholia.

Cases of *recurrent mania*—excluding alcoholic cases—have a persistent leucocytosis which rises gradually with the attack of excitement and falls again as the attack passes off. The polymorphonuclear percentage follows the curve of the hyperleucocytosis. Between the attacks the large lymphocytes and hyaline cells are increased, and may reach 20 per cent.

The leucocytosis of *acute continuous alcoholic mania* resembles that of acute continuous mania of non-alcoholic origin.

No leucocytosis was observed in the *delusional alcoholic cases*.

In *hebephrenia* the leucocytosis varies, the average being from 12,000 to 14,000; but a marked leucocytosis occurs now and again, the increase being chiefly due to the hyaline cells which may vary between 20 and 30 per cent.

During the period of recovery in *katatonia* a transient eosinophilia was observed.

Cases of delusional insanity exhibited no hyperleucocytosis.

At the onset of a typical case of *general paralysis* a hyperleucocytosis and a high polymorphonuclear percentage occurs. In the second stage the leucocytosis follows the course of the disease and is accentuated with the occurrence of a febrile attack. A transient eosinophilia is common during this stage. During the third stage the leucocytosis is irregular, the polymorphonuclear percentage may fall very low, and the lymphocytes are increased. If a marked remission should occur the leucocytosis falls to normal, but the polymorphonuclear percentage remains very low.

In *epileptic insanity* hyperleucocytosis is present, is persistent, and is most marked at the period of the seizures.

The leucocytosis of *puerperal insanity* does not differ from that of acute mania.

Striking differences were observed in the character of the individual cells: in young patients the polymorphonuclear cells were well formed, whereas in the old or debilitated cases these cells were small with ill-defined granules which stained badly. Degenerated and vacuolated cells were sometimes seen in acute and debilitated cases. Large lymphocyte cells—excluding the hyaline variety—are frequently increased, more especially in hebephrenia, katatonia, recurrent mania, and in debilitated cases.

H. DE M. ALEXANDER.

**COMPARATIVE PSYCHIATRY.** E. KRAEPELIN (of Munich), (317) *Centralbl. f. Nervenh. u. Psych.*, July 15, 1904, p. 433.

KRAEPELIN gives a short sketch of the results of his examination of the native patients in an asylum on Java. Many of the clinical types found in Europe were recognised among the natives, often modified by racial characteristics. On the other hand Kraepelin calls attention to the possibility of the existence of quite new types of insanity. Comparative psychiatry may be of the greatest help to comparative psychology.

C. MACFIE CAMPBELL.

## TREATMENT.

**THE TREATMENT OF NEURASTHENIA AND HYSTERIA BY**  
(318) **ISOLATION AND PSYCHOTHERAPEUTICS.** ANDRÉ  
THOMAS, *Presse méd.*, July 9, 1904, p. 435.

THIS short paper contains an interesting and masterly review of the subject, and an accurate analysis of the nature and general symptomatology of neurasthenia and hysterical functional ailments, though it has evidently been written only for the general practitioner.

The author draws attention to the progressive increase of the slighter functional nervous disorders, and the rarity to-day of "*la grande hystérie*," which, he remarks, belongs to another age. Neurasthenia, on the contrary, is "*la maladie de la mode*." It occurs to two forms, not, however, to be rigidly separated.

- (1) *constitutional*, where it chiefly asserts itself as a general asthenia in individuals unfitted, morally and physically, for normal life, who lack energy and resistance, initiation and reaction; and
- (2) *accidental*, often the result of some moral or physical shock,

there being, however, a definite disproportion between the cause and the effect. In the latter class there may be often a latent neurasthenic disposition, but the symptoms are generally concentrated to one or other organ.

It must be considered to which of these classes any one case belongs, but the general lines of treatment are the same in all. That recommended is that practised by Déjerine in the *Salpêtrière*. It consists in

1. Separation of the invalid from his or her family and ordinary surroundings, which, in the constitutional cases, have probably contributed to the development of the condition and in the accidental has often been the scene of the shock or direct causal factor.
2. Isolation, if possible, in a special building, where the patient cannot be in communication with his friends, either direct or by correspondence. It is necessary, too, that the patient should be separated from other such cases; it is the general experience that these cases do not improve satisfactorily in ordinary hospital wards. Such surroundings may excite the illness, or suggest its persistence or recurrence, while in strict isolation the invalid is spared the comparison with others. In isolation, too, the authority of the physician is greater, and the confidence of the patient is more easily gained.

Finally, while in isolation the patient has complete rest, and, under suitable diet (at first only milk in large quantities), his general nutrition rapidly improves.

3. Psychotherapeutics. The chief essential is that the patient has complete confidence in the physician and in his ability to cure. He must be persuaded and convinced of the nature of his illness. The disease is essentially an auto-suggestion, and the aim must be to scientifically re-educate the invalid, to reform and strengthen his character, to raise his physical, intellectual, and moral energy, and to restore to him the power of will, self-confidence, and the pleasure of existence.

Suggestion is, however, as a rule less successful in the neurasthenic than in the more hysterical (functional) cases.

GORDON HOLMES.

**REMOVAL OF THE SEMICIRCULAR CANALS IN A CASE OF (319) UNILATERAL AURAL VERTIGO.** RICHARD LAKE, *Lancet*, June 4, 1904, p. 1567.

THE case is that of a young woman, aged 21, who had suffered for five years from vertigo, tinnitus, and gradually increasing deaf-

ness. She was operated on after all the usual medicinal remedies had been tried in vain. The operation consisted of a mastoid exploration enlarged by means of a burr into the petrous temporal bone, so as to destroy the three semicircular canals in turn. After the operation the patient showed symptoms of considerable cerebral irritation till the seventh day, but subsequently the vertigo was entirely gone, and the hearing much improved, though the tinnitus continued as bad as before.

JOHN D. COMRIE.

**THE SURGERY OF IDIOCY AND INSANITY.** DA COSTA, *Journ.* (320) *Nerv. and Ment. Dis.*, June 1904, p. 386.

DA COSTA reviews the various surgical measures sometimes adopted in the treatment of idiocy and insanity. While acknowledging the propriety of surgical interference where distinct symptoms point to local brain trouble, he would greatly restrict the sphere of the surgeon as conceived by some. Surgery, for instance, is out of place in treating hypochondriacal delusions and hallucinations; a healthy organ should never be removed because visceral delusions exist. No operation is of use in the insanity that may accompany ordinary epilepsy. Operation is of no use in microcephalic idiocy, as Bourneville has conclusively shown that it is not caused by premature ossification of the skull.

Internal drainage for hydrocephalic idiocy and imbecility may ameliorate the condition; external drainage for any considerable time is dangerous. One should not operate on possible cases of traumatic insanity unless there be some definite indication of the seat of a lesion.

C. MACFIE CAMPBELL.

**TRANSPLANTATION OF TENDON FOR MUSCULO-SPIRAL PARALYSIS.** H. W. M. GRAY, F.R.C.S.Ed., *Lancet*, May 21, 1904, p. 1419.

GRAY records a case of successful transplantation of the flexor carpi ulnaris for musculo-spiral paralysis. The patient was a lad sixteen years old, who had suffered from a fracture of the humerus, followed by complete paralysis of the musculo-spiral nerve. Unsuccessful operations had been done to relieve this. Two years later, Gray transplanted the flexor carpi ulnaris by dividing its tendon just above the pisiform bone, pushing it through to the dorsal aspect of the wrist immediately above the upper border of the pronator quadratus, and uniting it to the distal end of the divided common extensor of the fingers. Three weeks later, when the dressings were removed, the boy was at once able to voluntarily extend his fingers at the metacarpo-phalangeal joint. It



was also found that the power of supination was markedly improved, so much so that Gray is of opinion that Tubby's plan in such cases of converting the pronator teres into a supinator, is unnecessary. Gray proposes to transplant the flexor carpi radialis into the extensors of the thumb at a later date.

J. W. STRUTHERS.

---

## Obituary

### KARL WEIGERT.

(The Editors' thanks for this Obituary Notice are due to Professor Ludwig Edinger of Frankfurt-on-Main.)

KARL WEIGERT died on the 5th of August, at Frankfurt-on-Main, in his fifty-ninth year, as the result of a thrombosis of the coronary arteries. No other living pathologist has exercised so great and so beneficent an influence upon his students. The broad, philosophical modes of thought which always led him to regard biological questions from a high point of view, the immense assiduity with which—more exact than most—he followed out his problems, the new questions which he raised and which he sought either by himself or through his pupils to answer, and above all the skill with which he treated problems of technique, have procured for Weigert a well-merited place of honour among the pathologists of our time. Religious prejudice—Weigert was a Jew by birth—denied to him a wider sphere of activity in a university; but to the small room in Dr Senckenberg's laboratory in Frankfurt there came a constant crowd of doctors from all countries, the highest honour that can be awarded to a scientific man.

To Weigert we owe the discovery of many entirely new facts. He first brought order into the greatly confused anatomical knowledge of nephritis; he explained the cases of sudden death from occlusion of the coronary arteries, and he discovered that miliary tuberculosis is always the result of the penetration somewhere of a tubercle into the blood-stream, and its subsequent dissemination. The most important problem which occupied him was that of the correlation of component parts. For Weigert there existed no "irritation" (Reiz); he saw only injury and over-growth of the normal cells in the affected regions. Parenchymatous inflammation was all he admitted; what others called interstitial was to him only secondary proliferation of the interstitial tissue. He investigated primary affection of the organic parts in the

nervous system, the liver, the kidneys, and many other regions, and he first gave to cirrhosis of the liver and to granular atrophy of the kidneys their proper place. It was not the connective tissue which compressed the cells, as had hitherto been believed, but alcohol, etc., that injured them, the affected tissue being replaced by connective tissue. This theory of destruction, which he alone discovered and upheld in conscious opposition to Virchow's teaching, he jestingly named after the Indian "god of destruction": the "Shiva-theory." From this he went on to set forth many new problems and to find the answers to them. Weigert was the first to maintain that all but the most serious injuries will be followed by a process of compensation. On this fact, which he firmly established, depends the well-known interpretation, first given by Ehrlich, of the condition of immune bodies, by which we first learned to understand how, for example, the introduction of diphtheritic-toxin into the tissue of an animal will be followed by the formation of antidiphtheritic-bodies.

To Weigert's technical skill we owe many new and most excellent methods of staining. He was the first to lay down the principle that a stain must be elective, that it must colour only certain parts of the tissue, leaving all others intact. The first fruit of such work was the staining of bacteria. What to-day is a simple and necessary technical process to every doctor, was till then an impossibility. Koch has repeatedly acknowledged what he owes in this respect to Weigert. To neurologists this has a special interest, as to Weigert we are indebted for the means of staining the medullary sheath, which first made it possible to investigate the structure of the normal organ, and which has led to so many pathological discoveries. All the modifications of his stain which came later have failed to improve on it; it is now used exactly as it was twenty years ago. By means of this method we first obtained a conception of the richness of the nervous apparatus. For the seventeen years preceding his death, Weigert was occupied with the problem of the glia with the elective stain with which he delighted the whole world, and which still remains the best, its discoverer alone was never satisfied. In his work since then Weigert occasionally found an object on which it was not successful—generally the brains of animals—and this kept him steadfastly at his work-table, pursuing through many years this self-set problem with a tenacity which often rendered him unhappy, inasmuch as the constant consideration of this one question prevented him from doing creative work in other regions, and left him time only for receptive work. Weigert read much; few men were so absolutely abreast of knowledge as he; especially was he ready to recognise with pleasure, and without a shade of envy, the work of others. He mastered all the languages of

culture in order to be able to follow the progress of science; he formed, both at home and when travelling, numerous relations with foreign scientists, English especially.

In the earnestness and industry with which he worked he was a pattern to all; he was absolutely absorbed in his work so long as the day lasted. But in the evening he became a charming, bright companion, a warm-hearted man to whom nothing was alien. He lived in close friendship with many of his students scattered over the world, receiving stimulation from them and communicating to them his enthusiasm. A visit to him was to them a special pleasure, and he received many such ever-welcome guests in his little study.

Science will go on progressing, even though this great man works no longer with her, but in the circle of his friends there will always remain a blank which it will be hard to fill.

# Bibliography

## ANATOMY

- ZUCKERKANDL. Über die Collateralfurche. *Arch. a. d. Neurol. Instit. Wien*, Bd. 11, 1904, S. 407.
- V. BIART. Fibrils and Ganglion Cells. *Med. Rec.*, Aug. 6, 1904, p. 217.
- HATSCHKE. Bemerkungen über das ventrale Haubenfeld, die mediale Schleife und den Aufbau der Brücke. *Arch. a. d. Neurol. Instit. Wien*, Bd. 11, 1904, S. 128.
- KARPLUS. Bemerkungen über die grauen Massen im Funiculus cuneatus der menschlichen Medulla oblongata. *Arch. a. d. Neurol. Instit. Wien*, Bd. 11, 1904, S. 171.
- E. ZUCKERKANDL. Die Riechstrahlung. *Arch. a. d. Neurol. Instit. Wien*, Bd. 11, 1904, S. 1.
- FRÖHLICH. Beitrag zur Kenntniss des intraspinalen Faserverlaufes einzelner hinteren Rückenmarkswurzeln. *Arch. a. d. Neurol. Instit. Wien*, Bd. 11, 1904, S. 378.
- J. P. KARPLUS u. A. SPITZER. Zur Kenntniss der abnormen Bündel im menschlichen Hirnstamm. *Arch. a. d. Neurol. Instit. Wien*, Bd. 11, 1904, S. 29.
- SPITZER. Über die Beziehungen der abnormen Bündel zum normalen Hirnbau. *Arch. a. d. Neurol. Instit. Wien*, Bd. 11, 1904, S. 55.
- IRVING HARDESTY. On the Development and Nature of the Neuroglia. *Am. Journ. of Anatomy*, Vol. iii., No. 3, 1904, p. 229.
- OBERSTEINER. Weitere Bemerkungen über die Fett-Pigmentkörnchen im Centralnervensystem. *Arch. a. d. Neurol. Instit. Wien*, Bd. 11, 1904, S. 400.

## COMPARATIVE ANATOMY—

- HERMANN DEXLER. Beiträge zur Kenntniss des feineren Baues des Zentralnervensystems den ungelagerten Gegenbauers. *Morpholog. Jahrb.*, Bd. 32, H. 2, 1904, S. 288.
- CAVALIÉ. Les ramifications nerveuses dans l'organe électrique de la torpille (*Torpedo galvanus*). *Bibliographie Anatom.*, T. xiii., 4<sup>e</sup> fasc., 1904, p. 214.
- E. RABAUD et R. ANTHONY. Étude anatomique et considérations morphogéniques, sur un exencéphalie proencéphale. *Bibliographie Anatom.*, T. xiii., 4<sup>e</sup> fasc., 1904, p. 188.
- POPPER. Ein Marsupialier-Rückenmark. *Arch. a. d. Neurol. Instit. Wien*, Bd. 11, 1904, S. 94.

## METHODS—

- ARTHUR P. HERRING. Clay Modelling in the Study of Anatomy. *Journ. of Am. Med. Ass.*, Aug. 13, 1904, p. 454.
- Mde. NABIAS. Nouvelle méthode de coloration rapide du système nerveux au chlorure d'or. *Bibliographie Anatom.*, T. xiii., 4<sup>e</sup> fasc., 1904, p. 221.
- JOHN TURNER. On the Primary Staining of the Rat's Brain by Methylene Blue. *Brain*, Spring, 1904, p. 64.

## PHYSIOLOGY

- VITTORIO SCAFFIDI. Über den feineren Bau und die Funktion der Hypophysis des Menschen. *Archiv. f. mikroskop. Anatom.*, Bd. 64, H. 2, 1904, S. 236.
- SANO. Les localisations des fonctions motrices de la Moelle. *Gaz des Hôpitaux*, août 1904, p. 916.
- ALBRECHT BETHE. Der heutige Stand der Neuronentheorie. *Deutsch. medicin. Wochenschr.*, Aug. 11, 1904, S. 1201.
- SILVESTRO BAGLIONI. Physiologische Eigenschaften der sensiblen und der motorischen Rückenmarkselemente. *Zeitsch. f. allgemein. Physiol.*, Bd. 4, H. 1, 1904, S. 113.

- M. S. TCHIRIEV. Propriétés électro-motrices du cerveau et du cœur. *Journ. de Physiol. et de Pathol. générale*, T. vi., No. 4, 1904, p. 671.
- H. BORUTTAU und F. W. FRÖHLICH. Erregbarkeit und Leitfähigkeit der Nerven. *Zeitschr. f. allg. Physiol.*, Bd. 4, H. 1, 1904, S. 153.
- GIULIO ANDREA PARI. Sull' allungamento riflesso dei muscoli dello scheletro. *Zeitsch. f. allg. Physiol.*, Bd. 4, H. 1, 1904, S. 127.
- WILFRED HARRIS. Binocular and Stereoscopic Vision in Man and other Vertebrates, with its Relation to the Decussation of the Optic Nerves, the Ocular Movements, and the Pupil Light Reflex. *Brain*, Spring, 1904, p. 107.
- EDWARD B. COBURN. Associated Movements of the Upper Eyelid and Lower Jaw. *Med. Rec.*, Aug. 13, 1904, p. 243.
- S. J. MELTZER and CLARA MELTZER AUER. The Effect of Suprarenal Extract upon the Pupils of Frogs. *Am. Journ. of Physiol.*, Vol. xi., Nov. 1904, p. 449.

### PSYCHOLOGY

- ARNETT. The Soul—A Study of Past and Present Beliefs. *Am. Journ. of Psychol.*, July 1904, p. 347.
- J. S. MACKENZIE. The Infinite and the Perfect. *Mind*, July 1904, p. 356.
- B. RUSSELL. Meinong's Theory of Complexes and Assumptions (11). *Mind*, July 1904, p. 336.
- MACDOUGALL. Facial Vision: a Supplementary Report, with Criticisms. *Am. Journ. of Psychol.*, July 1904, p. 383.
- WILHELM SPECHT. Über klinische Ermüdungsmessungen. *Archiv. f. die gesamte Psychologie*, Bd. 3, H. 3, S. 245.
- H. G. WELLS. Scepticism of the Instrument. *Mind*, July 1904, p. 379.
- F. H. BRADLEY. On Truth and Practice. *Mind*, July 1904, p. 309.
- T. M. FORSYTH. The Conception of Experience in its Relation to the Development of English Philosophy. *Mind*, July 1904, p. 394.
- PORTER. A Preliminary Study of the Psychology of the English Sparrow. *Am. Journ. of Psychol.*, July 1904, p. 313.
- JESSIE ALLEN. The Associative Processes of the Guinea-pig. A Study of the Psychical Development of an Animal with a Nervous System well medullated at Birth. *Journ. of Comp. Neurol. and Psychol.*, July 1904, p. 293.

### PATHOLOGY

- PIERRE MARIE et GEORGES GUILLAIN. Les dégénérationes secondaires du cordon antérieur de la moelle. *Rev. Neurol.*, juillet 30, 1904, p. 697.
- WM. G. SPILLER. Earlier Changes in Arteriosclerosis of the Nervous System. *Proc. of the Pathol. Soc. of Phila.*, May 1904.
- GEORGE LAMB and WALTER K. HUNTER. On the Action of Venoms of Different Species of Poisonous Snakes on the Nervous System. *Lancet*, Aug. 20, 1904, p. 518.
- IUMUCOPULO. Multiple Osteome des Gehirns. *Arch. a. d. Neurol. Instit. Wien*, Bd. 11, 1904, S. 89.
- D. J. M'CARTHY. Formation of Bone Tissue within the Brain Substance. A contribution to the inclusion theory of tumour formation. *Univ. of Penn. Med. Bull.*, Phila., May 1904.
- BUNZL. Zur Parasitologie des Gehirns. *Arch. a. d. Neurol. Instit. Wien*, Bd. 11, 1904, S. 156.
- ALZHEIMER. Einiges über die anatomischen Grundlagen der Idiotie. *Centralbl. f. Nervenheilk. u. Psychiat.*, Aug. 15, 1904, S. 497.
- THOMAS LEWIS. Note on a Case of Defective Development of the Lateral Cerebellar Lobes in a Dog. *Brain*, Spring, 1904, p. 84.
- MESSING. Drei Fälle von Porencephalie. *Arch. a. d. Neurol. Instit. Wien*, Bd. 11, 1904, S. 184.

### CLINICAL NEUROLOGY AND PSYCHIATRY

#### GENERAL—

- R. CLEMENT LUCAS. The Wightman Lecture on the Hereditary Bias and Early Environment in their Relation to the Diseases and Defects of Children. *Lancet*, July 30, 1904, p. 278.
- NEURATH. Die nervösen Komplikationen und Nachkrankheiten des Keuchstussens. *Arch. a. d. Neurol. Instit. Wien*, Bd. 11, 1904, S. 258

- M. ROTHMANN. Ueber die Bedeutung der Elektrodiagnostik und Elektrophotherapie in der modernen Medicin. *Deutsche Klinik*, 125 Lieferung, 1904, S. 287.  
 L. PIERCE CLARK. Notes on Some Uncommon Forms of Nervous Diseases. *Med. Rec.*, July 23, 1904, p. 121.  
 O. MAAS. Ueber ein selten beschriebenes familiäres Nervenliden. *Berlin. klin. Wchnschr.*, Aug. 1, 1904, S. 832.

#### MUSCLES—

- MÉRY. L'atrophie musculaire progressive d'origine myopathique. *Journ. des Praticiens*, août 20, 1904, p. 532.  
 J. A. ANDERSON. Ein Fall von Myotonie congenita. *Wien. med. Wchnschr.*, Aug. 7, 1904, S. 1544.

#### PERIPHERAL NERVES—

- J. H. LLOYD. Two Cases of Paralysis of the Ulnar Nerve: one following a severe attack of Typhoid Fever; the other caused by pressure during occupation. *Med. News*, June 18, 1904.  
 SCHULTZ. Über Fußlähmung, Speziell Peroneuslähmung, bei Rübenarbeitern. *Deutsch. Archiv. f. klin. Medizin*, Bd. 80, H. 5 u. 6, 1904, p. 520.  
 RAYMOND. Deux cas de lèpre nerveuse. *Archiv. de Neurol.*, août 1904, p. 97.  
**Polymyositis.**—L. HARRISON METTLER. Polymyositis. *Medicine*, Detroit, July 1904.  
 F. W. LANGDON. Multiple Neuritis: a Clinical Lecture. *Alienist and Neurologist*, St Louis, May 1904.  
 W. JESSEN u. E. EDENS. Polymyositis und Polyneuritis bei Morhillen. *Berlin. klin. Wchnschr.*, Aug. 8, 1904, S. 847.  
 AUERBACH. Zur Pathogenese der Polyneuritis. *München. med. Wchnschr.*, Aug. 16, 1904, S. 1470.  
**Radicular Palsies.**—R. T. WILLIAMSON. Paralysis from Lesion of the Upper Part of the Brachial Plexus. *Lancet*, Aug. 13, 1904, p. 447.  
 JOHN E. DONLEY. A Case of Uniradicular Brachial Palsy affecting the Fifth Cervical Nerve. *Boston Med. and Surg. Journ.*, Aug. 4, 1904, p. 133.

#### SPINAL CORD—

- Meningitis.**—LOUIS FISCHER. Cerebrospinal Meningitis, with remarks on its Diagnosis, Prognosis, and Treatment. *Med. Rec.*, Aug. 13, 1904, p. 245.  
 R. DEANE SWEETING. The Diagnosis of Epidemic Cerebro-Spinal Meningitis. *Dublin Journ. of Med. Sci.*, Aug. 1904, p. 101.  
**Tabes.**—JOHN PUNTON. Modern Views of Locomotor Ataxia. *Kansas City Med. Index-Lancet*, June 1904.  
 THOMAS et HAUSER. Les altérations du ganglion rachidien chez les tabétiques. *Nouv. Iconograph de la Salpêtr.*, mai-juin 1904, p. 207.  
 M. NONNÉ. Ein Fall von familiärer Tabes dorsalis auf syphilitischer Basis. Tabes bei der Mutter und ihren zwei hereditär-syphilitischen Töchtern. *Berlin. klin. Wchnschr.*, Jan. 1904, S. 845.  
 ANDRÉ LÉRI. Cécité et Tabes. J. RUEFF, Paris 1904.  
 ANDRÉ LÉRI. Le "Tabes-cécité." *Gaz. des Hôp.*, juillet 30, 1904, p. 845.  
**Myelitis.**—JAMES COLLIER. The Effects of Total Transverse Lesion of the Spinal Cord in Man. *Brain*, Spring, 1904, p. 38.  
**Pressure Paraplegia.**—WM. G. SPILLER and THEODORE H. WEISENBERG. Paraplegia Dolorosa caused by Vertebral Carcinomata, Spinal Caries, and Multiple Neuritis. *Univ. of Penn. Med. Bull.*, Phila., May 1904.  
**Poliomyelitis Anterior Chronica.**—CHARLES S. POTTS. Case of Progressive Spinal Muscular Atrophy in which the Atrophy began in the Extensors of the Hand and Fingers. *Univ. of Penn. Med. Bull.*, Phila., May 1904.  
**Syringomyelia.**—H. J. VETLESEN u. FRANCIS HARBITZ. Kasuistische Beiträge zur Lehre von der Syringomyelie. *Nord. med. Arch.*, Abt. 11, H. 1, 1904, S. 1.  
 CARLO GUALDI. Morbo di Dupuytren e Siringomieliia (Fine). *Rev. crit. di clin. Med.*, Luglio 30, 1904, p. 493.  
**Hæmatomyelia.**—LAIGNEL-LAVASTINE. Hæmatomyélie traumatique. *Nouv. Iconograph de la Salpêtr.*, mai-juin 1904, p. 240.  
**Subacute Combined Sclerosis.**—JAMES TAYLOR. A Case of "Subacute Combined Sclerosis," with profound anæmia. *Brain*, Spring, 1904, p. 27.

#### BRAIN AND MEMBRANES—

- Hydrocephalus.**—FUCHS. Ein Fall von sogenannten idiopathischen Hydrocephalus chron. internus (beim Erwachsenen) und Beitrag zur Lehre von den objektiven Kopfgerauschen. *Arch. a. d. Neurol. Instit. Wien*, Bd. 11, 1904, S. 62.

- Thrombosis.**—C. W. BURR and D. E. PFAHLER. Report of a Case of Cerebral Thrombosis. *Penn. Med. Journ.*, Pittsburg, June 1904.
- Hemiplegia.**—BRISSAUD et PÉCHIN. De l'hémiplégie oculaire. *Le Progrès Médical*, août 18, 1904, p. 97.
- Encephalitis.**—RAYMOND et CESTAN. L'encéphalite aiguë hemorrhagique. *Gaz. des Hôp.*, juillet 28, 1904, p. 829.
- W. P. CARR. Endematous Encephalitis. A study of some conditions found in operating for Cerebral Epilepsy and Allied Affections. *Internat. Journ. of Surgery*, New York, June 1904.
- Bulbar Paralysis.**—L. K. HIRSCHBERG. Pseudo-Bulbar (Glossio-Pharyngeal-Labial) Paralysis. *Med. News*, New York, July 9, 1904, p. 68.
- WILFRED HARRIS. Post-diphtheritic Chronic Bulbar Paralysis. *Lancet*, July 23, 1904, p. 209.
- PURVES STEWART. Unilateral Congenital Lesion of Medulla and Spinal Cord; Death from Pontine Hemorrhage. *Brain*, Spring, 1904, p. 89.
- Tumour.**—WM. H. WILDER. Eye Signs of Brain Tumour. *Chicago Med. Recorder*, June 15, 1904.
- CHARLES K. MILLS. Differential Diagnosis of Single or Multiple Brain Tumours and Diffuse Encephalic Syphilis. *Univ. of Penn. Med. Bull.*, Phila., May 1904.
- OTTO G. T. KILIANI. Some Remarks on Tumours of the Chiasm, with a proposal how to reach the same by operation. *Annals of Surgery*, July 1904, p. 35.
- WALTER D. BERRY. Intracranial Psammo-Sarcoma without Paralysis. *Am. Journ. of Insanity*, April 1904.
- F. E. WALKER. Sarcoma of the Brain. *Med. Herald*, St Joseph, Mo., June 1904.
- RENÉ CRUCHET. Tumeur caséuse du lobe gauche du cervelet. *Rev. Mens. des Malad. de l'Enfance*, août 1904, p. 349.
- Abscess.**—C. H. R. JORDAN. Report of a Fatal Case of Multiple Otitic Abscesses in the Temporal Lobe, with Remarks. *Archives of Otolaryngology*, June 1904, p. 186.
- Amaurotic Family Idiocy.** ERNEST SACHS. Case of Amaurotic Family Idiocy. *Kansas City Med. Index-Lancet*, June 1904.
- General Paralysis.**—JAMES MIDDLEMASS. Traumatism and General Paralysis. *Journ. of Ment. Sci.*, July 1904, p. 433.
- JOSEF A. HIRSCHL. Die Frühdiagnose der progressiven Paralyse. *Wien. med. Press*, Juli 24, 1904, S. 1462.
- SERVIGNY. La paralysie générale dans la Suisse romande. Etude statistique comparative sur la rareté de cette affection dans certaines contrées, spécialement dans le canton de Fribourg (suite et fin). *Annales Méd.-Psychol.*, juillet-août 1904, p. 48.
- JOHN KER. General Paralysis and Crime. *Journ. of Ment. Sci.*, July 1904, p. 437.
- Gunsshot Wound.**—SCHOTT. Zwei Fälle von Schussverletzungen des Gehirns. *München. medicin. Wchnschr.*, Aug. 9, 1904, S. 1432.

## GENERAL AND FUNCTIONAL DISEASES—

- LEONARD GUTHRIE. Neurotic Children. *Clin. Journ.*, Aug. 17, 1904, p. 285 (Concluded).
- L. HARRISON METTLER. Syphilis as a Cause of the Neuroses. *Chicago Medical Recorder*, May 15, 1904.
- H. M. DE JARNETTE. Headache, Neuralgia, Vertigo and Epilepsy in relation to Eye Strain. *Old Dominion Journ.*, Richmond, Va., June 1904.
- L. W. LITTIG. "Tics." *Iowa Med. Journ.*, Des Moines, June 15, 1904.
- A. L. BENEDICT. Oesophageal Neuroses. *Am. Journ. of Med. Sci.*, Aug. 1904, p. 327.
- COLMAN W. CUTLER. Migraine and Sick Headache. *Med. News*, July 30, 1904, p. 194.
- CHARLES H. BEARD. Eye Signs in Migraine. *Chicago Med. Recorder*, June 15, 1904.
- E. V. HUBBARD. Grave's Disease. *N.Y. State Journ. of Med.*, June 1904.
- W. CAMPBELL POSEY and WALTER C. SWINDELLS. Unilateral Exophthalmos in Exophthalmic Goitre; with report of a case. *Ophthalmic Record*, Chicago, May 1904.
- E. APERT. Myxoedème fruste, croissance tardive, diabète. *Nouv. Iconograph. de la Salpêtr.*, mai-juin 1904, p. 175.
- E. BRISSAUD et HENRY MEIGE. Type Infantile du Gigantisme. *Nouv. Iconograph. de la Salpêtr.*, mai-juin 1904, p. 165.

- WILLIAM W. GRAVES. Ueber Lückenbildung zwischen den einzelnen Zähnen, ein fruhdiagnostisches und bisher wenig bekanntes Zeichen der Akromegalie. *Monatschr. f. Psychiat. u. Neurol.*, Juli 1904, S. 18.
- J. N. HALL and C. E. WALBRACH. Adiposis Dolorosa, with Report of three cases. *Am. Journ. of Med. Sci.*, Aug. 1904, p. 313.
- W. B. WARRINGTON. Myasthenia Gravis with Ophthalmoplegia; with some observations on Ocular Palpiae. *Med. Chron.*, Aug. 1904, p. 319.
- AUBREY D. P. HODGES. Sleeping Sickness: A Resumé. *Lancet*, July 30, 1904, p. 290.
- J. P. L. HULST. Beitrag zur Kenntniss der Fibrosarkomatose des Nervensystems. *Virchows Archiv*, Bd. 177, H. 2, Aug. 1904, p. 317.
- F. RUDLER. Un cas de neurofibromatose généralisée. *Nouv. Iconograph. de la Salpêtr.*, mai-juin 1904, p. 203.
- Epilepsy.**—E. SAVARY PEARCE and L. NAPOLEON BOSTON. The Blood in Epilepsy; Experiments on Animals. *Am. Journ. of Insanity*, Baltimore, April 1904.
- L. PIERCE CLARK and THOS. P. PROUT. Status Epilepticus. *Am. Journ. of Insanity*, April 1904.
- F. E. COULTER. Epilepsy—Diagnosis and Treatment. *Western Med. Rev.*, Lincoln, Neb., June 1904.
- B. SACHS. The Relation of Epilepsy, Chorea, and other Motor Disturbances of the Nervous System to Eye Disease. *Med. News*, July 30, 1904, p. 197.
- MARC RAY HUGHES. Psychical Epileptic Equivalence. *Med. Mirror*, St Louis, May 1904.
- Paralysis Agitans.**—WM. G. SPILLER. Points of Resemblance between Paralysis Agitans and Arthritis Deformans. *Univ. of Penn. Med. Bull.*, Phila., May 1904.
- Chorea.**—L. SCHAPS. Blutbefunde bei Chorea minor und Tic convulsif. *Jahrb. f. Kinderheilk.*, Juli 1, 1904, S. 29.
- JAMES T. WRIGHTSON. A Unique Case of Chorea. *New York Med. Journ.*, July 23, 1904, p. 158.
- CARL D. CAMP. Paralytic Chorea, with a report of two cases. *Univ. of Penn. Med. Bull.*, Phila., May 1904.
- Hysteria.**—SEYMOUR J. SHARKEY. Hysteria and Neurasthenia. *Brain*, Spring 1904, p. 1.
- B. SIDIS and MORTON PRINCE. Contribution to the Pathology of Hysteria based on an experimental study of a case of Hemianesthesia with chronic convulsive attacks simulating Jacksonian Epilepsy. *Boston Med. and Surg. Journ.*, June 23, 1904.
- L. HARRISON METTLER. Differential Diagnosis between Hysteria and Neurasthenia, and Treatment. *Med. Standard*, Chicago, July 1904.
- J. J. MOREN. Hysterical or Functional Paralysis. *Louisville Monthly Journ.*, Louisville, Ky., June 1904.
- MERZBACHER. Kasuistische Beiträge zur hysterischen Artikulationsstörung speziell des hysterischen Stotterns. *München. med. Wchnschr.*, Aug. 18, 1904, S. 1468.
- Tetany.**—RUDOLF FUNKE. Über Pseudo-tetanie. *Prager med. Wchnschr.*, Nr. 33, 1904, S. 432. (Fortsetzung aus Nr. 29.)
- T. GILLMAN MOORHEAD. A Case of Gastro-Tetany. *Practitioner*, Aug. 1904, p. 160.
- ALCOHOLISM, POISONS, ETC.**—
- W. C. SULLIVAN. A Statistical Note on the Social Causes of Alcoholism. *Journ. of Ment. Sci.*, July 1904, p. 417.
- PELLETIER. Die psychologischen und sozialen Ursachen des Alkoholismus. *Med. Blätter*, Aug. 4, 1904, S. 379.
- W. L. HOWARD. Dipomania and its Treatment. *Med. News*, Aug. 6, 1904, p. 264.
- ROBERT JONES. The Relation of Inebriety to Insanity, and its Treatment. *Lancet*, Aug. 6, 1904, p. 368.
- J. M. BUCHANAN. The Treatment of the Morphine Habit by Hyoxin. *Am. Journ. of Insanity*, April 1904.
- E. KNECHT. Zur Kenntniss der Erkrankungen des Nervensystems nach Kohlenoxydvergiftung. *Deutsch. Med. Wchnschr.*, Aug. 18, 1904, S. 1242.
- MENTAL DISEASES.**—
- HANS BERGER. Experimentelle Studien zur Pathogenese der Geisteskrankheiten. *Monatschr. f. Psychiat. u. Neurol.*, Juli 1904, S. 1.



- KUHLMANN. Experimental Studies in Mental Deficiency: Three cases of Imbecility (Mongolian) and six cases of Feeble-mindedness. *Am. Journ. of Psychol.*, July 1904, p. 391.
- A. SCHOTT. Beitrag zur Lehre von der Ideenflucht in der Manie. *Centralbl. f. Nervenh. u. Psychiat.*, Aug. 15, 1904, S. 519.
- FRIEDMANN. Löwenfeld's Buch über die psychischen Zwangsercheinungen. *Centralbl. f. Nervenh. u. Psychiat.*, August 15, 1904, S. 506.
- F. QUENSEL. Neue Erfahrungen über Geistesstörungen nach Schwefelkohlenstoffvergiftung. *Monatschr. f. Neurol. u. Psychiat.*, Juli 1904, p. 48.
- CHARLES L. DANA. Eye Strain and the Psychoses. *Med. News*, July 30, 1904, p. 193.
- RICHARD DEWEY. Syphilis as a Cause of Psychoses. *Chicago Medical Recorder*, May 15, 1904.
- OTTO FOHN. Some Metabolic Studies, with special reference to Mental Disorders. *Am. Journ. of Insanity*, April 1904.
- I. H. CORIET. Chemical Findings in the Cerebro-Spinal Fluid and Central Nervous System in various Mental Diseases. *Am. Journ. of Insanity*, April 1904.
- L. C. BRUCE and A. S. M. PEEBLES. Quantitative and Qualitative Leucocyte Counts in various forms of Mental Disease. *Journ. of Ment. Sci.*, July 1904, p. 409.
- E. MARANDON DE MONTYEL. La prédisposition en étiologie mentale. *Journ. de Neurol.*, juillet 5 et 20, 1904, p. 241.
- W. R. DUNTON, Jr. Report of a Second Case of Dementia Præcox, with Report of Autopsy. *Am. Journ. of Insanity*, April 1904.
- L. TREPSAT. Un cas de démence précoce catatonique avec pseudo-œdème compliqué de purpura. *Nouv. Iconograph. de la Salpêtr.*, mai-juin 1904, p. 193.
- A. PICK. Über primäre progressive Demens bei Erwachsenen. *Prager med. Wchnschr.*, Aug. 11, 1904, S. 417.
- DENY. Les déments vésaniques. *Gas. des Hôpitaux*, août 1904, p. 913.
- HENRY R. STEDMAN. A Case of Moral Insanity, with repeated Homicides and Incendiarism and late Development of Delusions. *Boston Med. and Surg. Journ.*, July 21, 1904, p. 57.

## SPECIAL SENSES AND CRANIAL NERVES—

- WARD, A. HOLDEN. The Eye Symptoms of Hysteria, Neurasthenia and the Traumatic Neuroses. *Med. News*, July 30, 1904, p. 200.
- GRASSET. La deviation conjuguée des yeux de l'hémianopsie. *Rev. Neurol.*, juillet 15, 1904, p. 645.
- KNAPP. Hereditary Optic Atrophy. *Archives of Ophthalmology*, July 1904, p. 888.
- MAXIMILIAN BONDI. Über hysterische Augenmuskel-Lähmungen in einer Familie. *Prager med. Wchnschr.*, Nr. 33, Aug. 15, 1904, S. 429.
- W. CAMPBELL POSEY. Palsy of the Extra-ocular Muscles in Exophthalmic Goitre. *Am. Journ. of Med. Sci.* Philadelphia, June 1904.
- H. GRADLE. Paralysis of the Associated Lateral Eye Movements, presumably due to Acute Polio-encephalitis Superior, ending in Recovery. *Archiv. of Ophthalm.*, New Rochelle, N.Y., May 1904.
- E. L. OATMAN. Hysterical Iridoplegia and Cycloplegia, with Report of a Case. *Archiv. of Ophthalm.*, New Rochelle, N.Y., May 1904.
- CHARLES A. OLIVER. Clinical and Histologic Study of the Ophthalmic Condition in a case of Cerebellar Neoplasm occurring in a Subject with Renal Disease. *Am. Journ. of Med. Sci.*, Philadelphia, June 1904.
- C. H. FRAZIER. Surgical Treatment of Facial Palsy. *Penn. Med. Journ.*, Pittsburg, June 1904.
- DOPTER. Paralyse faciale, ourlienne, lymphocytose du liquide céphalo-rachidien. *Gas. des Hôp.*, août 2, 1904, p. 857.
- RAYMOND. Diplegie faciale, syphilis et grande hystérie (suite et fin). *Journ. des Practiciens*, août 13, 1904, p. 518.
- ALEXANDER. Zur chirurgischen Behandlung der peripheren Facialis-Lähmung: Pfropfung des Facialisstammes an den Nervus hypoglossus. *Archiv. f. Ohrenheilk.*, Bd. 62, H. 1 u. 2, 1904, S. 1.
- GEORGE HEATON. The Symptoms and Treatment of the Intra-cranial Complications of Otitis Media. *Lancet*, July 30, 1904, p. 292.
- ALEXANDER and V. FRAENKL-HOCHWART. Ein Fall von Acousticus-tumor. *Arch. a. d. Neurol. Inst. Wien*, Bd. 11, 1904, S. 885.
- SOUQUES. Des troubles auditifs dans les tumeurs cérébrales. *Rev. Neurol.*, juillet 30, 1904, p. 727.

## MISCELLANEOUS SYMPTOMS—

- G. MARINESCO. Recherches sur la sensibilité vibratoire. *Presse Méd.*, août 13, 1904, p. 513.  
 PAUL SAINTON et ROGER VOISIN. Contribution à l'étude du trophœdème. *Nouv. Iconograph. de la Salpêtr.*, mai-juin 1904, p. 189.  
 LOEPER et CROUZON. Contribution à l'étude des hémicédèmes chez les hémiplegiques. *Nouv. Iconograph. de la Salpêtr.*, mai-juin 1904, p. 181.  
 E. MASING. Sprachstörungen. *St Petersburg med. Wchnschr.*, Aug. 1904, Sn. 321 u. 333.  
 TREITEL. Ueber Störungen des musikalischen und Sprachgehörs. *Deutsche med. Wchnschr.*, Juli 28, 1904, S. 1134.  
 DEJERINE et THOMAS. Un cas de cécité verbale avec agraphie suivi d'autopsie. *Rev. Neurol.*, juillet 15, 1904, p. 655.

## TREATMENT—

- SANGER BROWN. Hypodermic Injection of Strychnine Nitrate in the Treatment of Progressive Muscular Atrophy. *Med. Rec.*, Aug. 6, 1904, p. 204.  
 ALTSCHUL. Ueber Suggestivtherapie bei funktioneller Neurose. *Prager med. Wchnschr.*, Aug. 4 u. 11, 1904, S. 404.  
 CHARLES W. BUCKLEY. Observations on the Treatment of Neurasthenia. *Practitioner*, Aug. 1904, p. 176.  
 ERNST BARTH. Ein neuer Gesichtspunkt bei der Behandlung der Aponia Spastica. *Deutsch. med. Wchnschr.*, Aug. 4, 1904, S. 1174.  
 WM. B. SNOW. The Employment of Static Electricity in the Treatment of Nervous Diseases. *Journ. of Advanced Therapeutics*, N.Y., June 1904.  
 HENRY RAYNER. Need of Care of the Weak-minded in Infancy and Childhood. *Journ. of Ment. Sci.*, July 1904, p. 473.  
 CONOLLY NORMAN. On the Need for Family Care of Persons of Unsound Mind in Ireland. *Journ. of Ment. Sci.*, July 1904, p. 461.  
 J. CARLYLE JOHNSTONE. The After Care of Friendless Patients discharged from Asylums as provided for by the Scottish Probationary Procedure. *Journ. of Ment. Sci.*, July 1904, p. 455.  
 D. G. THOMSON. A Few Remarks on the Registration of Nurses and the Nurses Registration Bill from the Mental Nursing Point of View. *Journ. of Ment. Sci.*, July 1904, p. 451.  
 GEORGE P. LOVE. Surgical Treatment of the Insane. *Toledo Med. and Surg. Reporter*, June 1904.  
 B. M. RICKETTS. Surgery of Hydrocephalus. *Am. Med.*, June 18, 1904.  
 E. P. FRIEDRICH. Ueber die chirurgische Behandlung der otogenen eitrigen Cerebrospinal Meningitis. *Deutsch. med. Wchnschr.*, Aug. 4, 1904, S. 1167.

# Review of Neurology and Psychiatry

## Original Article

### **A FURTHER STUDY ON THE SENSORY SEGMENTAL ZONE OF THE UMBILICUS.**

By WILLIAM G. SPILLER, M.D.,

Professor of Neuropathology and Associate Professor of Neurology in the  
University of Pennsylvania, and

T. H. WEISENBURG, M.D.,

Instructor in Nervous Diseases and in Neuropathology in the  
University of Pennsylvania.

From the Philadelphia General Hospital.

From the Wm. Pepper Laboratory of Clinical Medicine (Phoebe A. Hearst  
Foundation).

Read before the Philadelphia Neurological Society, April 26, 1904.

THE exact determination of the sensory zone in which the umbilicus lies is of great importance as regards the surgery of the spinal column. In a paper read by one of us (Spiller) before this Society, Dec. 23, 1901, and published in the *Philadelphia Medical Journal*, Feb. 8, 1902, the different views held by investigators of this subject were presented. It may be well to refer to these briefly, in order to show the doubt prevailing in regard to the sensory zone of the umbilicus.

Boettiger thinks it is uncertain whether the umbilicus lies in the distribution of the ninth or tenth thoracic segment; Walton places it in the distribution of the eleventh thoracic segment; Dejerine in the distribution of the tenth thoracic segment; Head regards the tenth thoracic sensory segment as in the subumbilical region and the upper border of this segment

as passing directly through the umbilicus; Wichmann believes that the umbilicus lies in the area of the tenth thoracic segment; Paterson places it between the tenth and eleventh thoracic segments; Kocher at the lower border of the tenth thoracic segment; and Seiffer draws a line from the umbilicus a little downward, and then upward and around the trunk, and regards this as the limit of the tenth thoracic segment.

From this review of the literature it will be seen that there is much to be said in favour of the situation of the umbilicus within the tenth thoracic sensory segment, but this is an opinion we are unable to accept.

In the paper by one of us (Spiller) referred to above, a case was reported which seemed to show that the umbilicus lies between the ninth and tenth thoracic sensory segments. The line defining the anæsthetic area passed directly through the umbilicus, and the tenth thoracic segment of the cord was much softened. The ninth thoracic segment was believed to be intact, but further study shows it is possible that the lower part of this segment was affected, so that instead of the umbilicus lying exactly on the border between the ninth and tenth thoracic sensory segmental areas, we may conclude from this case that it may lie in the lower part of the ninth thoracic sensory segmental zone. The correction is not very important.

We have had the opportunity to confirm the views formed from a study of this case. A man was struck in the back by the falling of a bank of earth, and was brought to the Polyclinic Hospital, Nov. 17, 1899. He had complete paralysis of both lower limbs, and loss of sensation below a line drawn around the abdomen about one inch below the umbilicus, resulting from fracture of the vertebræ. Operation was performed by Dr John B. Roberts, and the displaced fragments of bone were removed. The patient was transferred to the Philadelphia General Hospital, Oct. 8, 1900. He remained completely paralysed in his lower limbs until his death in the summer of 1903. The lower limbs were much atrophied, he had severe bedsores, the patellar and Achilles reflexes and skin reflexes of the lower limbs were lost, and no movement of the toes was produced by plantar irritation. He had incontinence of urine and fæces and a girdle sensation. Sensation in all forms was lost in both lower limbs and over the abdomen to a line drawn one inch below the umbilicus, even

until the examination made shortly before the patient's death. This line was sharply defined.

A careful microscopical examination of sections taken from the upper part of each segment of the spinal cord from the tenth thoracic to the upper part of the cervical enlargement showed that as high as the upper part of the tenth thoracic segment the cord was completely destroyed. Many normal fibres entered the cord in the posterior roots of the ninth thoracic segment.

This examination seems to show conclusively that the umbilicus, in this case at least, was not situated in the tenth thoracic sensory segmental zone, inasmuch as the tenth thoracic segment of the spinal cord was entirely destroyed, and the zone of anaesthesia ceased one inch below the umbilicus. The umbilicus therefore probably lies within the ninth thoracic sensory segmental zone, and this conclusion is in conformity with the findings of the previous case. The importance of this determination must be apparent on account of the prominence of the umbilicus as a surgical landmark.

In this case, as in the former case, the Babinski reflex was not obtained, and in both cases the sacral region of the cord was softened. The absence of this reflex in these cases of lesions of the central motor tracts may be explained by destruction of the reflex arc, and it would be well if more attention were paid to the condition of this reflex in complete transverse lesions of the cord above the lumbar region. It is possible that in this sign we may have a means of judging of the condition of the sacral region of the cord in cases of fracture of the vertebrae.

---

## Abstracts

### ANATOMY.

**A CONTRIBUTION TO THE STUDY OF THE FINER STRUCTURE (322) OF THE RETINA.** GUIDO SALA, *Anatom. Anzeiger*, Bd. xxv., 1904.

THIS is a short paper describing and figuring certain appearances observed by Dr Sala in the retinae of rabbits, dogs and cats, when stained by Cajal's method. The inter-nuclear layer is found to consist mainly of cells with large nucleus and nucleolus and

numerous branching processes forming a complex meshwork. The protoplasm, both of the cells and the processes, has a finely fibrillary structure. The relation of the ends of the processes to the capillaries is striking, the fine ends of the processes often encircling the capillary in a sort of spiral.

Dr Sala is not sure whether these cells are to be looked upon as essentially supporting cells, or as true nerve cells. He maintains that careful observation shows that the processes never really anastomose, though imperfect observation is apt to mislead on this point. The peculiar relationship of the ends of the processes to the small blood-vessels has, he thinks, some connection with the function of these processes. Fine illustrations of the appearances described are appended to the article.

J. V. PATERSON.

**A STUDY OF THE ABNORMAL BUNDLES IN THE HUMAN (323) BRAIN-STEM.** J. P. KARPLUS and A. SPITZER, *Arch. a. d. Neurol. Instit. Wien*, Bd. xi., 1904, S. 29.

In this article a careful description is given of the course of a bundle of fibres arising in the lower pontine region and descending to lose itself in the medulla.

The material was taken from an apparently normal brain and serial sections were stained by the Weigert-Pal method from the lower part of the pons to the upper limit of the pyramidal decussation.

The bundle is first seen originating as a compact mass of fibres in the lowest part of the pons. It emerges from among the crossing fibres of the latter, courses in a dorso-lateral direction, crossing the emerging seventh nerve; then, separating itself from the rest of the pontine fibres, it passes in a dorsal direction through the corpus trapezoides to reach the tegmentum; in this course it divides into two main bundles, which these further subdivide, all of the groups of fibres passing caudalwards to disappear in the medulla. In this course a cross section shows the various bundles lying chiefly in the grey matter at the base of the fourth ventricle, central to the fasciculus solitarius.

Considering how closely associated in their origin are these fibres with the ncl. acustic., the dorsal ncl. of vagus, the course of the spinal root V, of the glossopharyngeal and vestibular roots, the authors conclude that these bundles probably represent a central sensory path passing upward to end either in the pons, cerebellum, or cerebrum.

According to the authors, "Pick's bundle" consists of two masses of fibres: one descending, consisting of descending pyra-

midal fibres as described by Hoche; and a second ascending, i.e. the fibres described in this paper.

The article is well illustrated by many plates and an excellent diagram of the course of the fibres. STANLEY BARNES.

**ON THE RELATIONSHIP OF THE ABNORMAL BUNDLES TO**  
 (324) **THE NORMAL BRAIN STRUCTURE.** A. SPITZER, *Arch. a. d. Neurol. Instit. Wien*, Bd. xi., 1904, S. 55.

COLLECTING together the evidence with regard to Pick's bundle and similar "abnormal" medullary tracts, the author discusses whether they should be considered as abnormal, and if so, in what way. He concludes that the abnormality only consists in the fibres being collected together to form a compact bundle, and that scattered fibres representing the same function are present in every medulla. He regards the collection of fibres into compact tracts as part of a phylogenetic process, the odd outlying fibres often representing a far more scattered distribution at an earlier stage of phylogenetic development. The descending fibres of Pick's bundle he regards as the remains of the upper part of the pyramidal decussation, which at one time was far more widely distributed over the medulla than now. STANLEY BARNES.

**A CONTRIBUTION TO THE COURSE AND TERMINATION OF**  
 (325) **GOWERS' TRACT.** J. RAMSAY HUNT, *Pub. of Cornell Univ. Med. Coll.*, Vol. i., 1904, p. 19.

In the case here described the cord was pressed on by tuberculous caries and pachymeningitis at the level of the seventh cervical segment. The consequent ascending and descending degenerations were studied by Marchi's method. The chief interest of the case is in connection with the course of Gowers' tract. The author's observations are in agreement with those of Bruce, Winter, and Dydynski, for in his case the tract "curved backward at the trigeminus level through the anterior medullary velum towards the cerebellum," and "no degenerations were observed anterior to the posterior corpora quadrigemina." Previous observations regarding the anatomy of this tract are summarised.

EDWIN BRAMWELL.

**THE INTRA-SPINAL COURSE OF CERTAIN POSTERIOR ROOTS**  
 (326) **OF THE SPINAL CORD.** A. FRÖHLICH, *Arch. a. d. Neurol. Instit. Wien*, Bd. xi., 1904, S. 378.

SOME of the lower cervical and upper dorsal posterior roots of an ape were cut in their intra-dural course. Stained by the Marchi

method, many degenerated fibres were found in the dorso-lateral part of the direct cerebellar tract above this region, and were traced up to the first cervical segment.

The author discusses whether these fibres are of posterior root origin, or whether they are degenerated as a result of injury to the direct cerebellar tract at the time of operation. No definite conclusion is arrived at.

STANLEY BARNES.

**THE OLFACTORY RADIATIONS.** E. ZUCKERKANDL, *Arch. a. d.* (327) *Neurolog. Institut. Wien*, Bd. xi., 1904, p. 1.

THERE is no difference of opinion about the course of the lateral olfactory radiations; it is generally accepted that they are centripetal tracts to the lobus piriformis and thence to the cornu ammonis and fascia dentata. But there has been, on the other hand, great divergence as regards the course and constitution of the medial radiations which connect the Gyrus fornicatus, G. subcallosus, cornu ammonis and fascia dentata with the olfactory lobes, corpus mamillare, uncus and tuberculum olfactorium. This has been so great, even from the experimental side, that Zuckerkandl has decided to tackle the subject again with Marchi's method, and precedes it with the following description of the topography of the tracts as they are seen by Weigert's stain.

The fornix longus is a complex system of fibres which come from the gyrus fornicatus and the cornu ammonis. Some of them pass through the corpus callosum, others through the psalterium. It is not a compact bundle, but a scattered layer of fibres as it enters the septum. That part of it to which the name has been reserved by most authors runs along the ventral surface of the corpus callosum and with the fasciculus subcallosus enters the olfactory lobe. Part of the remainder joins the fornix and the fasciculus hippocampi.

The bulk of the fibræ perforantes psalterii enter the fasciculus hippocampi, partly as precommissural fibres, while others join the fornix. The cingulum, which runs along the dorsal surface of the corpus callosum and turns ventralwards round its genu, also enters the septum intermingled with the more oral perforating fibres of the corpus callosum. These fibres may in part enter the pedunculus olfactorius and tuberculum olfactorium.

It is of considerable bulk in the lower mammals where the dorsal part of the cornu ammonis is well developed, but is small or absent in the higher, where this is reduced to the gyrus supracallosus.

Part of the fasciculus hippocampi passes caudalwards on the



base of the brain and ends in the corpus mamillare, basal nucleus (of Ganser) and uncus.

The fasciculus proprius olfactorius runs in front of the tuberculum olfactorium ; its relations are not definitely determined.

GORDON HOLMES.

**REMARKS ON THE GREY MATTER OF THE FUNICULUS  
(328) CUNEATUS OF THE HUMAN MEDULLA OBLONGATA.**

J. P. KARPLUS, *Arch. a. d. Neurolog. Institut. Wien*, Bd. xi., 1904, p. 171.

THE relation of the grey matter of the spinal trigeminus root (substantia gelatinosa Rolandi) to the dorsal column nuclei has not hitherto been properly described. It has been frequently confused with the lateral division of the nucleus funiculi cuneati which Clarke first figured, but can easily be distinguished from this, and from the rest of the nucleus, by its relatively smaller cells and by the fact that in contrast to these it contains very few medullated fibres.

Part of the substantia gelatinosa Rolandi lay within the funiculus cuneatus or its nucleus in every one of the twenty-six series examined by the authors, but the arrangement is very variable and inconstant and often asymmetrical.

GORDON HOLMES.

**REMARKS ON THE VENTRAL TEGMENTUM REGION, THE  
(329) MEDIAN FILLET AND THE STRUCTURE OF THE PONS.**

R. HATSCHKE, *Arch. a. d. Neurolog. Institut. Wien*, Bd. xi., 1904, p. 128.

THE author starts with a fairly complete review of our knowledge of the structures which lie in the tegmentum of the mid-brain between the superior cerebellar peduncles and the fillet, but has to remark how incomplete it is despite the number of facts which can be collected from comparative studies.

*Ventral tegmental commissure.*—This is the first structure to which he draws attention. In some animals, as in Phoca and the mouse, its fibres form a large and compact mass, but in others they are rather diffuse. They cross the middle line as a commissure, not as a decussation, ventral to the decussatio brachii and separated from it by the nucleus centralis superior. None of its fibres can be traced further cerebralwards, but caudalwards they lie on each side dorsal to the fillet and can be followed as far as the motor nuclei of the trigemini. He has been unable to discover anything

more as to their relations, but thinks he can exclude any connection with the brachia, the fillets, the central trigeminal tracts, or with other known tracts in this region.

*Corpus parabigeminum*.—Its development is very variable in the several classes of mammals. One bundle of fibres passes dorsalwards from it to the tectum, another medialwards across the median fillet, and a third ventro-caudalwards to the pontine ganglia. Its connections are not definitely known, but it seems to belong to the tectum, receiving afferent tracts (probably from the tractus spino-tectalis) and giving origin to others.

*Median fillet*.—In almost all animals it is devisable in its course through the pons and mid-brain, into a medial and a lateral portion, whose relative sizes vary very considerably. It is found that in animals in which the fore-extremities are well-developed the lateral division is large; while, contrariwise, when the forelimbs are small, as in the dolphin, it is absent or relatively very feeble. From these facts it may be concluded that fibres from Goll's nucleus form the median and those from Burdach's the lateral portion. This arrangement has already been described by Bechterew from his study on the myelinisation of the fibres.

*Pons varolii*.—The development of this region is also very variable throughout the mammalian classes. In the monotremata, for instance, the pyramidal tract and the fillet can scarcely be separated, while in other genera, as in man, they lie far apart. This is in main due to the development of the pontine nuclei and its deeper transverse fibres.

From comparative observations, Hatschek seems able to conclude that the stratum superficiale is the phylogenetically older system and that its development only bears relation to that of the cerebellum, while the strata complexum and profundum, and the pontine grey matter develops parallel with the cortico-pontine systems. The cerebello-fugal fibres belong to the former, the cerebello-petal are contained in the latter strata.

GORDON HOLMES.

**THE TRUE FORM OF THE BRACHIAL PLEXUS AND ITS  
(330) MOTOR DISTRIBUTION.** WILFRID HARRIS, *Journ. Anat.  
and Physiol.*, July 1904, p. 399.

HARRIS has dissected the brachial plexus in thirty bodies with a view to ascertaining the exact motor distribution of the various roots entering the plexus. His paper is divided into four sections: the first dealing with the fifth cervical nerve and its connections, the second with the connections between the outer and inner cords, the third with the posterior cord, and the fourth with the differences between the prefixed and post-fixed types of plexus.

As the result of his observations he has determined that the plexus in man is of the prefixed or high type as compared with that of monkeys below the anthropoids, for the higher roots at any rate, and that therefore the results of stimulation experiments in monkeys cannot be accepted as indicating the distribution of the nerves in the human subject. He has found by careful dissection several communications between the outer and inner cords not generally referred to, notably a branch to the ulnar nerve from the outer cord, forming an "outer head" for the ulnar. This was present in twenty-six of his thirty cases. He has also found that in a large proportion of cases the first dorsal nerve sends a branch to the posterior cord, although the branch from the lowest trunk of the plexus to the posterior cord is usually described as derived from the eighth cervical nerve only. Basing his conclusions partly on the results of stimulation of some of the cervical nerves in four operation cases, partly on two cases of lesion of the fifth cervical nerve, and partly on his dissections, Harris has drawn up lists of the motor supply of the muscles for the pre- and post-fixed types of plexus. His lists differ considerably from those of Starr and Edinger, and Poirier and Charpy, with regard to some of the scapular, upper arm and forearm muscles.

J. W. STREUTHERS.

**ON THE PRIMARY STAINING OF THE RAT'S BRAIN BY  
(331) METHYLENE BLUE.** JOHN TURNER, *Brain*, Spring 1904,  
Vol. xxvii., pp. 64-83.

RECORDS the successful application to the rat's brain of the author's method of methylene blue and peroxide of hydrogen staining, which hitherto has proved refractory to all but the human brain.

The method, which is very uncertain in its results, seems to depend upon the due development of a lactic acid fermentation in the brain.

The results obtained (in cortex cornu ammonis and striate body) are similar to those in man, but demonstrate the presence of the "dark" cells in both first and second layer of cortex, in which situations they have not been shown by this method in man.

These "dark" cells are regarded by the writer as an intermediary system interposed between the end afferent stations and first efferent (*e.g.* in the cortex), and as also occurring between afferent stations of different levels (*e.g.* Clarke's column—Optic Thalamus), and will in all probability be met with between the different stations on the efferent side, both cerebral cerebellar (and caudal).

They are distinguished from the pyramidal (or pale) system by four characteristics: (1) *dark staining*; (2) *shape*—very rarely pyramidal, usually square, pentagonal, or oval; (3) *character of dendrites*, which spring abruptly as slender branches from the cell body and run very long distances without sensible alteration in calibre, presenting an irregular contour with moniliform swellings or lateral offshoots and which pursue a *sinuous* course, whereas the dendrites of the pyramidal cells usually run rectilinearly; (4) *absence of definite orientation*.

It is contended that these cells give off from their dendrites extremely fine beaded fibres which form a loose network over the pyramidal cells, and that the dark cells are all in organic continuity with each other by means of this network.

Attention is also called to results sometimes obtained by subjecting the brain to a temperature between 60° and 100° C. whilst in the stain, whereby glial elements are picked out and the nervous largely neglected.

The glial cells stain nearly black, are much smaller than the nervous and Golgi nets, are distinctly seen, and it can be demonstrated that they arise from the ramifications of the glial cell branches. In the human cerebellum many of the cells of the granule layer are shown to be more or less immature glia cells.

AUTHOR'S ABSTRACT.

## PHYSIOLOGY.

### THE NEURONE THEORY AND ITS PRESENT POSITION.

(332) A. BETHE, *Deutsche med. Wchnschr.*, Aug. 11, 1904, p. 1201.

BETHE summarises the chief arguments against the neurone theory, and explains how, from being one of its supporters, he has come to be strongly antagonistic to it. The theory in its original form includes the following theses:—Each neurone is a developmental unit, arising from a single embryonic cell. The neurone is also an anatomical unit, comprising ganglion cell, dendrites, and axis-cylinder, all of these being parts of the one cell. No nerve units exist apart from neurones. The neurones are in contact, but not in continuity, one with another, the dendrites and nerve terminations ending blindly. The neurone cell, moreover, is regarded as a trophic and a functional unit.

Bethe discusses these various propositions in detail, and demonstrates their untenability. Amongst the most damning facts against the neurone theory, he emphasises the fact of regeneration of peripheral nerves independent of the ganglion cell, and the un-

doubtedly multicellular origin of the peripheral nerve fibre. It is highly probable that other nerve elements do exist, genetically independent of the neurones. Both in invertebrates and vertebrates there is a true anastomosis between different nerve cells in the fine nerve network of the grey matter, and fibrils can be traced from one neurone into another. Nor can the neurone any longer be considered as a trophic or functional unit, inasmuch as degenerative changes following various lesions are not restricted to the neurone primarily affected.

The name of "neurone" may still be of use, if it is restricted simply to a concept consisting of ganglion-cell, dendrites, and axis-cylinder. The term, however, should be reserved merely as a convenient diagram or scheme for teaching purposes. The essential elements are not neurones, but neurofibrillæ, which traverse the neurones and unite the whole nervous system into one vast anastomosing network.

PURVES STEWART.

**ON THE PRESENT POSITION OF THE NEURON THEORY. E.**  
(333) LUGARO, *Archiv. di Anat. e di Embriol.*, Vol. iii., f. 2, 1904.

IN this paper, Lugaro weighs the existing evidence in favour of and against the various doctrines that constitute the neuron theory. At the outset he insists upon the right of any scientific theory to undergo evolution with the elucidation of new facts bearing upon it. He warmly protests against the unnecessary vehemence of language and the hasty conclusions of some of those who have opposed the neuron theory, and charges them with having erroneously regarded scientific theories as beliefs incapable of being modified in the slightest degree without being wholly destroyed. He points out that, as a matter of fact, the neuron theory, which existed long before it received its name, has been undergoing a gradual process of evolution up to the present time. It may have to be further modified as the result of recent observations such as those of Apathy and Bethe, but there is still much difference of opinion as to the actual facts and many important points have not yet been made clear. If we ought to be ready to revise a theory in the light of new facts, it is also necessary that we should submit new observations to critical examination before we accept them.

The neuron theory comprises several fundamental principles intimately related to each other, but not so essentially connected that the destruction of one entails the like fate of the others. It is necessary to examine the various fundamental principles separately and to see in how far the new observations are in harmony with them, and to what extent they impose modifications upon

them. These fundamental principles are four in number:—(1) The neuron is an anatomical unity. (2) One neuron is connected with another by contact and not by continuity. (3) Physiologically the cell-body and the dendrites are organs of reception, whilst the axone is the organ of discharge (law of dynamic polarisation). (4) The nerve cell is the trophic centre of its prolongations.

The principle of anatomical unity of the neuron is conceivable in two senses. In a narrower sense we may regard it as implying that the neuron is a cellular unity; in a wider sense, we may conceive of the neuron as an organic individuality, as a complete organ, leaving unprejudiced the question of its embryological origin and of its cellular unity. The author examines the existing evidence in favour of the opposing views regarding the origin of the peripheral nerve fibres, dealing especially with the controversy between Bethe and Münzer as to the question of autogenous regeneration. He concludes that the matter cannot yet be regarded as settled. If, however, the pluricellular origin of the nerve fibres were to be fully demonstrated, the neuron theory would not be destroyed. The conception of the neuron as a cellular unity would merely have to give place to that of the neuron as an organic unity. He next examines some of the evidence in support of the conclusions of Apathy, Bethe and Nissl as to the continuity of neuro-fibrils, and contends that even if it were satisfactorily demonstrated that there is a continuity of the fibrils from one nerve cell to another, the conception of cellular unity would not necessarily be destroyed, and that of organic unity would certainly not. He concludes that whilst the view of the neuron as a cellular unity is at present controvertible, the conception of it as an anatomical or organic unity is not so.

Dealing next with the question of whether there is continuity or contact of neuro-fibrils, Lugaro admits the validity of Apathy's evidence in support of continuity in certain invertebrates, but denies that it is permissible on the ground of this evidence to formulate the same conclusion with regard to vertebrates. There are abundant data which tend to show that such a conclusion is erroneous. He discusses the question of the nature of the pericellular network of Golgi, which some observers have regarded as a nervous structure, and maintains his former opinion that it is an artificial product. He defends as positive evidence of discontinuity the histological pictures given by Golgi's method, which the opponents of the neuron theory have endeavoured to explain away, and attaches great importance to the recent demonstration by Cajal of terminal enlargements of the neurofibrils where they are applied to the surface of the nerve-cells and dendrites. He concludes that continuity has not been demonstrated in vertebrates, and that it is probable that continuity in invertebrates is a special

case, a special adaptation to peculiar functional requirements not existing in vertebrates.

As regards the law of dynamic polarisation, he finds nothing that is essentially opposed to it in the results of the experiment of Bethe upon *carcinus maenas*. They consist with this law as modified some years ago by Cajal. The nerve current in the dendrites does not always pass in a cellulipetal direction; it is axipetal, and thus in cases in which the axone is given off from a dendrite it may not pass through the cell-body. The conclusion of Bethe regarding the independence of the neurofibrils, which does not consist with this law, is opposed by the recent positive observations of Cajal and Donaggio.

Lastly, after examining the bearing of the recent experimental observations of Bethe upon the question, he concludes that even if we regard as demonstrated the pluricellular origin of the nerve fibres and the autogenous regeneration of nerves, the law of Waller still holds as the law of nervous trophism in the adult organism.

W. FORD ROBERTSON.

**SECONDARY DEGENERATIONS OF THE ANTERIOR COLUMN  
(334) OF THE CORD (THE DIRECT PYRAMIDAL FASCICLE  
AND THE CROSSED PYRAMIDAL FASCICLE. THE PARA-  
PYRAMIDAL TRACTS OF THE ANTERIOR COLUMN).**  
PIERRE MARIE and GEORGES GUILLAIN, *Rev. Neurol.*, July 30,  
1904.

THE work is a continuation of the study, the results of which were published by MM. Marie and Guillain in the *Semaine Medicale*, 1903, p. 19, and it is specially a reply to two stringent criticisms which have been passed upon the earlier publication, the one by Ugoletti, *Rivist. d. Pat. nerv. e. mentale*, April 1903, and the other by M. and Mme. Dejerine, *Rev. Neurol.*, 1904, p. 253.

The writers of this work contend that the ventral pyramidal tract has little, if any, of its origin in the cerebral cortex; and that in following the degenerations resulting from even the most extensive cortical lesions, by the Weigert method, they were unable to confirm the results of Van Gehuchten, Edinger, Obersteiner, Sharpy and others, that a direct pyramidal tract of cortical origin occupies the inner portion of the ventral column of the spinal cord. They found either a total absence of degeneration in this position, or very slight sclerosis confined to the cervical region. On the other hand they found that very definite degenerations of the ventral columns of the cord result from peduncular lesions.

They conclude that the bulk of the so-called direct pyramidal tract is composed of "para-pyramidal" fibres having their origin

presumably in the pedunculo-pontal region, and not in the cerebral cortex. Ugolotti criticises the results of MM. Marie and Guillain thus:—He states that there is no difference in the aspect of the degenerated direct pyramidal tract corresponding with a cortical and with a brain stem lesion. He thinks that the variations in the situation of the degeneration found by Marie and Guillain resulted from the examination of sections at different levels of the spinal cord, and finally he declares that all methods of tracing degenerations are fallacious except the method of Marchi. In reply, Marie and Guillain say that while they admit the superiority of Marchi's method, it is utterly illogical to exclude results obtained by Weigert's method, especially as this is the only method applicable to cases of long standing lesion. M. and Mme. Dejerine are of opinion that the results of Marie and Guillain are not justified by facts, and they deny that the direct pyramidal tract comprises an encephalic and a mesencephalic bundle. They state that the bulbar pyramid contains nothing but fibres of cortical origin, and they explain the results they criticise on the ground of the well-known variability of the direct pyramidal tract. They further exclude all cases of brain stem lesions where the lesion involved the tegmental region, since tracts entering the ventral column of the cord having their origin in the tegmentum may be degenerate (tecto-spinal and ponto-spinal descending systems). Marie and Guillain, in reply to the Dejerines, hold to their original opinions. They think that the exclusion of cases where the lesion implicates not only the pyramid but also the tegmentum vexatious, since in their original communication they stated their opinion that some of the "para-pyramidal" fibres coursed through the tegmentum.

They analysed in detail the cases which the Dejerines have published in their confutation, and come to the conclusion that these cases supported rather than negated their opinion.

They proceed to bringing forward further evidence as follows:—

Eight cases of extensive cortical and cortico-central lesions examined by Weigert's method. The direct pyramidal degeneration was absent in five cases, and very slightly marked in three cases.

Five cases of cortical and cortico-central lesions examined by the method of Marchi. Slight degeneration in the direct pyramidal tract only was found.

One case of a large pontine lesion examined by Weigert's method. Extensive degeneration in both ventral columns and both crossed pyramidal tracts.

A case of recent softening involving the right peduncle, completely destroying the right red nucleus and the adjacent tegmentum, Marchi method. Marked degeneration in the right



ventral column of the cord and right post-longitudinal bundle, central tegmental tract, and left rubro-spinal tract. A case of recent softening involving the left peduncle, and similar in details to the above.

In these two cases the so-called para-pyramidal fibres are figured in the position of the crossed rubro-spinal tract (Monakow's bundle), while there is copious degeneration in both posterior longitudinal bundles.

The authors proceed to state their opinion that the degenerations in the ventral column of the cord are much more extensive in lesions of the brain stem than in the lesions of the cortex, and that such difference is to be explained not by variations in the decussations of the pyramids, but on the ground of fibres having their origin in the brain stem and descending in the tegmentum to enter the ventral column of the cord below the pyramidal decussation. These are the para-pyramidal fibres.

The reviewer's opinion is that the deductions of MM. Marie and Guillain are quite unjustifiable and entirely at variance with pathological and experimental evidence, and is based upon the grounds:—

(1) Most extensive degeneration throughout the length of the ventral column of the cord occurs in some cases of purely cortical lesions (Marchi method).

(2) It is impossible to follow with any accuracy by Weigert's method degenerations of scattered fibres in regions of mixed origin.

(3) If the fibres described by Marie and Guillain are not fibres of—

1. The rubro-spinal tract (Monakow's bundle).
2. The tecto-spinal tract (ventral longitudinal bundle).
3. The dorsal longitudinal bundle.
4. A descending thalamo-spinal tract, described by Collier and Buzzard in the cat.

MM. Marie and Guillain have not proved this point.

(4) If the "para-pyramidal" fibres are identical with the above, it is difficult to perceive why the authors should name them "para-pyramidal," their only connection with the pyramidal fibres being that they enter the same region of the cord as the minority of the pyramidal fibres.

JAMES COLLIER.

#### **A PSEUD-AFFECTIVE REFLEX AND ITS SPINAL PATH.**

(335) R. S. WOODWORTH and C. S. SHERRINGTON, *Journ. of Physiol.*, Vol. xxxi., p. 234.

If in the cat under deep chloroform narcosis the cerebral hemispheres and thalamencephalon be removed, on the applica-

tion of peripheral stimulation after relaxing the administration of the anaesthetic certain motor reactions can be observed. Among these *pseud-affective* reflexes are some mimetic movements simulating the expression of certain psychical states, e.g. turning of the head and neck toward the point stimulated, vocalisation angry or plaintive, etc., etc. In the normal animal these movements are taken as the outward sign of a concurrent inward feeling. The removal of the brain in front of the mesencephalon annihilates the neural mechanism which renders the inward feeling possible, but it leaves fairly intact the reflex motor mechanism through which the outward expression is manifested. When the expression occurs, it is assumed that, had the brain been present, the feeling would have occurred. These *pseud-affective* reflexes the authors have utilised in attempting to determine the spinal path conducting those impulses that they assume would have evoked pain if the brain had been intact. A spinal lesion which prevents the occurrence of the expression in response to a stimulus that previously excited the expression has been regarded by them in their experiments to be such as would induce analgesia in regard to that stimulus had the brain been present. Their method was to prepare, by means of the above reaction, the effect of two stimuli symmetrically applied on opposite sides of the body, after semisection or other lesion of the cord in front of the nerve fibres stimulated.

After semi-section of the cord at the level of the thirteenth thoracic segment, the reaction was obtained by stimulation of either sciatic nerve or any of its main branches (purely cutaneous—internal saphenous—or purely muscular—hamstring), but more vigorously and promptly from the nerve on the same side as the lesion, and from this nerve also the reaction was evoked by weaker stimulation. This would indicate that the pathway taken by afferent impulses eliciting expressions of pain is, from the hind limb, both crossed and uncrossed, but more largely crossed. The same was found on stimulation of the brachial nerves or their branches after semi-section of the cord at the third or fourth cervical segment, only the difference was more marked here than between the two sides of the body in the hind limb region. The headward algesic path from the arm is, therefore, in the anterior-cervical region mainly crossed. Stimulation of the splanchnics on the two sides after semi-section of the cord in the anterior cervical region led to a similar result. From all these unilateral regions, therefore, the afferent *pain* path in the cord is bilateral, the larger share belonging to the crossed side.

With regard to the question as to which of the columns of the cord convey these impulses, the authors were able to exclude the dorsal and ventral columns, and the central grey matter. They

must therefore pass through the lateral columns. This they proved by direct experiment as well as by a process of exclusion.

They come to the conclusion, (1) "that the *lateral* column furnishes the headward path in the spinal cord for *nociceptive* (algesic) arcs; (2) that each lateral column conveys such impulses from *both* lateral halves of the body, and somewhat preponderantly those from the crossed half; and (3) that this is true for these arcs whether they be traced from *skin*, *muscle*, or *viscus*."

SUTHERLAND SIMPSON.

**FURTHER OBSERVATIONS ON THE FAT PIGMENT GRANULES  
(336) IN THE CENTRAL NERVOUS SYSTEM.** OBERSTEINER,  
*Arch. a. d. Neurol. Institut. Wien*, Bd. xi., 1904.

THE author in this paper—a continuation of previous work—while recognising that the light yellow nerve-cell pigment is a physiological product of cell metabolism, maintains that it undergoes pathological alterations. He cites two examples which he has found in the same case: an aggregation of the pigment around the nucleus (peripheral lipolysis), and a reticular formation of it which suggests an intermediate stage in the irregularly proceeding depigmentation of the cell. Excess, or complete absence of the pigment, are pathological.

DAVID ORR.

## **PATHOLOGY.**

**THE CHANGES IN THE SPINAL ROOT GANGLIA OF TABETICS.**  
(337) THOMAS and HAUSER, *Nouv. Icon. de la Salpêtrière*, May-June 1904.

THESE authors have studied the chronic changes in the root ganglia of tabetics by a method specially adapted to show the gross lesions.

In the various cases examined there were several inconstant features, but a certain degree of cell destruction was evident in every case.

There were many degrees of nerve cell atrophy and much evidence of cell destruction, many areas showing distinct paucity of cells lying adjacent to other areas in which the nerve cells were fairly well preserved.

The highest degree of cell change was associated with advanced atrophy of the posterior roots. In all cases the lumbar and sacral ganglia were more affected than the dorsal, and these in turn more

than the cervical. Naturally, nerve cell destruction was most evident in the more chronic cases.

The position formerly occupied by nerve cells was sometimes indicated by concentric nuclear proliferations derived from the cell capsules, but at other places cell destruction was followed by fibrous or hyaline changes in the connective tissue. Many atrophied cells were not surrounded by proliferated nuclei, from which fact the authors conclude that cell atrophy and capsular proliferation are two phenomena which do not have any causal relationship. The capsular proliferation may either be primary and inflammatory, or secondary in order to absorb the debris of nerve cells.

As to whether the ganglion cell changes are secondary to the atrophic changes in the roots is discussed in the light of the new results obtained after division of these, but Thomas and Hauser have not come to any definite conclusion as yet.

Although the nerve cell changes in the root ganglia of tabetics are so marked, the authors are forced to conclude that, in spite of their frequency, it is difficult to appreciate what rôle they play in the pathogenesis of the root and cord degenerations.

They are too frequent, however, and so marked in certain cases not to play some part in the general pathogenesis of tabes.

DAVID ORR.

#### **OBSERVATIONS ON THE ANATOMICAL BASIS OF IDIOCY.**

(338) ALZHEIMER, *Centr. f. Nervenheilk. und Psychiat.*, Aug. 15, 1904.

SCARCELY any other category, says the author, has played a greater rôle in the recent development of psychiatry than that of secondary dementia. This term, which originally included all states of acquired mental enfeeblement, is to-day falling into desuetude, and becoming split up into its various forms—dementia præcox, alcoholic, epileptic and paralytic dementia, and so on. Idiocy, however, corresponds in many directions to dementia, and is undergoing in the same way a division into classes sharply distinguished from each other. That is the symptomatological is giving place more and more to the clinical classification. Here, however, a difficulty is encountered, for in the case of idiocy the disease affects an incomplete brain and hinders its development, giving to the clinical pictures so many features in common that the distinguishing characteristics are obscured. Their solution, therefore, must rest ultimately on their pathological anatomy.

The author then discusses the various states met with in idiot brains—macro- and microcephaly, macro- and microgyrie, porencephaly, etc—and shows that any one of these states may be the

result of very different conditions, various destructive processes, or arrested development. The first form considered is that of "paralytic" idiocy, in the author's experience frequently encountered amongst congenital idiots and imbeciles. The paralytic condition, however, has not, in his opinion, existed from birth in many of these cases, but has been grafted on to a congenital defect, and a short account is given of two cases in which he was able to demonstrate, besides the cortical changes characteristic of general paralysis, changes depending upon vascular lesions, and producing an arrest of development at an earlier life period. With these "graft-paralyses" also he classes the cases described by Bourneville as meningitic idiocy. The consideration also of other classes of idiocy, *e.g.* that of amaurotic idiocy, described by Warren, Tay and Sachs, a disease severely "galloping" in course, and leading to death in the course of a few months, has led to the discardure of the idea, maintained formerly by many—*e.g.* Ziehen—that in idiocy one had to do with a completed and unprogressive condition. A further example is furnished by that of the hypertrophic tuberos scleriosis of idiots, cases of which have been described by Bourneville, and in Germany by Brückner, and which, like the last, is a progressive disease, developing, it may be, till the tenth year, or even—if, as the author inclines to believe, Fürstner's cases belong also to this category—not fully developing till later life.

Formerly, in the etiology of idiocy, arrested development of the brain took a very important place, and it was attempted to establish a parallelism between the degree of development of the idiot brain and that of a normal brain at an earlier period. According to the author's experience, such parallelism is rarely found, and in the great majority of cases various morbid conditions, degenerated nerve-cells, sclerotic changes and diseased vessels are met with. The probable causes of these conditions are then summed up, some cases being perhaps a particularly early and severe form of epilepsy, others, as Kraepelin has suggested, an early form of dementia præcox. It is certain, however, says the author, that under the conditions met with in idiocy there are concealed diseases which are only early or particularly severe forms of morbid processes encountered also in the adult.

R. CUNYNGHAM BROWN.

#### COMPENSATORY PROCESSES IN THE HUMAN SPINAL CORD.

(339) A. PICK, *Neurol. Centralbl.*, July 16, 1904, p. 641.

PICK figures the spinal cord from a case of old infantile hemiplegia, in which the posterior cornu on the side of the pyramidal sclerosis was broader than on the normal side throughout the whole extent of the cord. This anomaly was most marked in the cervical and

lumbar regions, but was also recognisable in the thoracic region. He considers the enlargement of the posterior cornu to be a true hypertrophy which had occurred at a period when it still had the power of growth; in fact, he regards it as compensatory.

PURVES STEWART.

**THE EFFECTS OF TOTAL TRANSVERSE LESION OF THE  
(340) SPINAL CORD IN MAN.** JAMES COLLIER, *Brain*, Spring  
1904, p. 38.

THIS paper is based upon a large number of personal observations. Details of fifteen cases, with nine autopsies, are given. In two cases operative procedure allowed the determination of the nature of the lesion.

Evidence is brought forward that as the result of total transverse lesion of the spinal cord in man, the knee-jerks and other deep reflexes are permanently abolished in the region supplied by the caudal segment. Further, as time elapses after the occurrence of a total lesion, the muscles waste and slowly lose their faradic excitability, and the sphincters lose their tone, the only sign of self-action remaining on the isolated part of the spinal cord being the occasional presence of certain of the skin reflexes (plantar reflex) in much reduced degree. These phenomena occur in the absence of any recognisable structural change in the ventral horn cells, ventral roots and peripheral nerves of the paralysed region.

Attention is drawn to the fact that when the knee-jerks have disappeared, in cases of progressive transverse lesions becoming total, for a certain time after such disappearance, the knee-jerks may be again obtained subsequent to long-continued faradisation of the lower limbs. The lumbo-sacral centres in man have not sufficient self-action to maintain the muscle tone when severed from the higher centres, but they may be temporarily roused by peripheral stimulation into such a higher state of activity as will allow the knee-jerk to be obtained. The importance of this fact here recorded for the first time in proving that the loss of knee-jerk is due to isolation from the higher centres, and not to any concomitant injury to, or consecutive changes on, the lumbo-sacral centres, is obvious.

Referring to the results of total transverse section in animals, it is shown that the capacity for self-action in the isolated portion of the spinal cord diminishes from the lower to the higher mammals, and is least in man. Dr Sherrington has shown that in monkeys transverse section is followed by results closely resembling those which obtain in man. The author would explain the persistence of the knee jerk in several authentic

cases of total transverse lesion in man as depending upon an exceptional individual peculiarity of the spinal cord in these cases reverting to the condition obtaining in the monkey, the "spinal animal" capacity in these subjects being much greater than in the average man. Hence, the loss of knee-jerk from total lesion is the rule in man, its persistence the rare exception. The writer has never met with a case of total transverse lesion of the spinal cord with persistent knee-jerks.

He points out that in many of the cases which have been published as examples, the totality of the lesion has not been proved.

He considers that a total lesion is only proved:—

1. When the spinal cord is actually severed.
2. When serial longitudinal sections through the region of the lesion stained by Weigert's method show no myelinated fibres passing through the length of the lesion, and when special methods for staining axis cylinders show a similar absence of these structures in the length of the lesion.
3. When serial transverse sections through the region of the lesion, stained by a nuclear stain show the whole area of transverse section to be necrotic.

Physiological total lesion may be considered to exist when there is absolute loss of sensibility, complete flaccid paralysis; and loss of the deep reflexes.

He submits that the return of the knee-jerks at a long interval after the occurrence of a total transverse lesion may be due to the re-establishment of conducting paths by regeneration. Two of the cases recorded support this opinion.

The order of appearance of the symptoms where gradual lesions progress to totality is given, and also the order of regression where a physiologically total lesion is recovered from.

The condition of the sphincters and of the skeletal muscles in the paralysed region which obtained in the writer's cases are described in detail.

The results are widely at variance with those obtained by experiment upon animals, progressive loss of the sphincter tone and progressive wasting of the muscles with lowering and finally loss of faradic excitability having occurred.

AUTHOR'S ABSTRACT.

**ON THE PARASITOLOGY OF THE BRAIN.** VICTOR BUNZL,  
(341) *Arch. a. d. Neurol. Instit. Wien*, Bd. xi., 1904.

THROUGHOUT the brain of a common mole (*Talpa europæa*) numerous spirally coiled and encapsuled nematodes were found. The parasites, measuring about  $16\mu$  in diameter and approximately

200 $\mu$  in length, resembled the embryos of *Trichina spiralis*. Their identity could not, however, be definitely determined.

W. T. RITCHIE

### CLINICAL NEUROLOGY.

**ON NEUROFIBROMATOSIS.** JOSEPH FRAENKEL and J. RAMSAY (342) HUNT, *Pub. of Cornell Univ. Med. Coll.*, Vol. i., 1904.

NEUROFIBROMATOSIS is defined by the authors as the formation of one or more tumours in one or more cerebrospinal or sympathetic nerves.

The tumours usually originate from the endoneurium, but may arise from the peri or epineurium. Their congenital origin has been demonstrated in a large number of cases, while associated stigmata of degeneracy are frequent. Five clinical types may be distinguished:—

- (1) Tubercula dolorosa.
- (2) Multiple fibromata of the skin (molluscum fibrosum, Recklinghausen's disease).
- (3) Neuromata (cranial or spinal) or neurofibromata of a single peripheral nerve.
- (4) Neuroma plexiforme, pachydermatocele, and elephantiasis neuromatodes.
- (5) Generalised neurofibromatosis; neurofibromata of the skin, cerebrospinal and sympathetic nerves.

The pathological and clinical characters of these various types are mentioned, and in conclusion the history of four cases narrated.

Case 1. Compression myelitis, produced by intervertebral fibroma.

Case 2. Isolated neuromata of the posterior tibial and communicans-peronei nerves. Hyperæsthesia, paralysis, operation, recovery.

Case 3. Type of Recklinghausen's disease. Fibromata of peripheral nerves. No symptoms.

Case 4. Multiple skin fibromata. Few nerve fibromata. Symptoms doubtful.

EDWIN BRAMWELL

**POLYMYOSITIS AND POLYNEURITIS AFTER MEASLES.** (343) JESSEN and EDENS, *Berl. klin. Wchschr.*, Aug. 8, 1904.

IN the first case, that of a woman æt. 32, on the tenth day after the eruption of the measles rash, a polymyositis, first of the left, then of the right leg, occurred. The symptoms were classical: great pain on active, less on passive movement; tenderness over the whole of the affected limb, along with increase in the volume and great hardness of the muscles, and cedema of the dorsum of the foot. The right leg was attacked a week after the left, just as



the polymyositis in the latter was subsiding. The polymyositis was accompanied by fever, but by no enlargement of the spleen or affection of the mucous membranes. Recovery was complete.

The nervous complications of measles are usually either cerebral or spinal, neuritis being much more rare. The patient was a girl, *æt.* 16, who was attacked by measles, and during the course of the disease suffered from suppurative otitis media and bronchopneumonia. On the ninth day after the rash she complained of great pain and tenderness in the back and legs, which ultimately reached an extreme degree of hyperæsthesia and spread to the right arm. As the sensory disturbance grew less, paresis of the legs was observed, along with tenderness over the course of the larger nerve trunks. Eventually the neuritis subsided entirely, and the patient regained complete health in about six weeks from the onset of the measles.

J. S. FOWLER.

#### ON A SPECIAL FORM OF FAMILY NEUROTIC MUSCULAR

(344) **ATROPHY (DÉJÉRINE-SOTTAS).** MARTIN BRASCH, *Deutsch.*

*Zeitschr. f. Nervenh.*, Band 26, Heft 3, S. 302.

A FULL but concise family history is given of a condition occurring in three generations, characterised by club-foot (in the first), by club-foot and an additional symptom-complex (in the second and third), and associated (in another member of the third generation) with progressive paralysis. The clinical picture of the father and son (second and third) shows the onset of the disease, at about the age of thirteen years, by a weakness in the legs, advancing rapidly to double club-foot (*equino-varus*), and presenting (before the age of forty) a characteristic Aran-Duchenne muscular atrophy in the arms and hands. In addition were noted fibrillary twitchings in the affected muscles, slight loss of sensation in the extremities of the limbs, absence of tendon-reflexes, diminution of electrical excitability in nerves and muscles, with no sphincter involvement. So far the condition corresponds exactly with the progressive neurotic muscular atrophy of Hoffmann.

But the patients betrayed further myosis, absence of light reflex, Rombergism and ataxia in a most pronounced degree. The natural deduction of an accidental (or otherwise) combination of two apparently distinct diseases in the same individual is negatived, according to the author, by the exact resemblance of his cases to two (brother and sister) described by Déjérine and Sottas in 1893. The same possibility had struck these latter observers, the atrophic and the ataxic elements forming so prominent a feature of the condition. Post-mortem examination, however, revealed marked thickening of the peripheral nerves (this had been easily palpable

*intra vitam*), whence the nomenclature, "progressive hypertrophic neuritis."

The pathology of the condition being still somewhat uncertain, the writer is content to observe in conclusion that the amyotrophy of Charcot-Marie cannot be limited to the narrow description of its first observers, but that the group of cases beginning with those of Déjérine and Sottas do form a clinical, if not a pathological entity.

There are references to the literature.

S. A. KINNIER WILSON.

**A CASE OF MID-DORSAL POTTS' DISEASE, COMPRESSION-**  
(345) **MYELITIS, RECOVERY.** J. RAMSAY HUNT, *Pub. of Cornell Univ. Med. Coll.*, Vol. i., 1904, p. 18.

A MAN, aged 35, of good family history, who had been operated on for a tuberculous testicle. Difficulty in walking developed, and in six months paralysis was complete, with severe rigidity and some amount of anaesthesia.

The bladder, anal and genital functions were conserved but weakened. Rest in the recumbent position for a considerable time had no effect. Dr Elliot applied a plaster jacket; complete recovery within about a year.

AUTHOR'S ABSTRACT.

**A CASE OF SYRINGOMYELIA AND SYRINGOBULBIA, WITH**  
(346) **DEGENERATION OF THE LEMNISCUS.** S. A. KINNIER WILSON, *Rev. de Méd.*, Sept. 10, 1904, p. 685.

DETAILS are given of a case diagnosed as syringomyelia during life in which there was little sign of definite involvement of the medulla. The autopsy revealed a characteristic syringomyelic cavity in the cord, extending from the first cervical to the fifth dorsal segment. In addition, the pathological process had involved the medulla from just above the pyramidal decussation to the level of the inferior olives in their upper part. The cavity was irregularly sinuous, stretching across from the head of one olive to that of the other. The bulbar pyramids were practically non-existent, except for one or two isolated groups of fibres, chiefly external. Above the cavity the pyramidal paths showed marked retrograde degeneration. The microscopical examination is discussed in detail.

The cord showed marked pyramidal degeneration at all levels, but there was no sign whatever anywhere of degeneration in the direct pyramidal tracts. The relation of these to the main paths is analysed.

The lemniscus on the right side was fuller than that on the left, which was shorter, slimmer, and more pointed posteriorly. At the same time, its coloration with hæmatoxylin was less in-

tense. This asymmetry of the fillet was limited to its inferior extent, between the posterior column nuclei and the upper levels of the olives. The whole constitution of the sensory paths in the medulla, with the question of the localisation of fibres or groups of fibres in them, is examined, and the conclusion is reached that probably the asymmetry represents a lesion in fibres of short trajectory, passing from the nucleus of Burdach's column on the right to the left inferior olive, which is more destroyed than its fellow of the opposite side.

There are references to the literature.

AUTHOR'S ABSTRACT.

**ON A RARE FAMILY NERVOUS DISEASE.** O. MAAS, *Berl.*  
(347) *klin. Wchnschr.*, Aug. 1, p. 832.

THE writer describes in great detail the cases of a brother aged 29 and sister aged 26, children of a drunken father and a mother who suffered from senile dementia. In the brother, weakness began in the legs at 13 and spread later to the arms; atrophy of both upper and lower extremities was marked, the thenar and hypothenar eminences being almost gone; fibrillary tumours were visible in limbs and tongue; the facial muscles also weak; the knee-jerks much increased and the plantar reflex extensor in type; no interference with sensory or visceral functions; intelligence good. In the sister's case, weakness commenced at 12; her condition of combined atrophy and spasticity was much the same as the brother's; in addition the speech was blurred and she was decidedly weak-minded. In both cases the *fundus oculi* was normal.

The writer mentions four similar cases from one family recorded by Hoffmann, who ascribed the condition to amyotrophic lateral sclerosis. He also refers to similar cases recorded by Seligmüller and Gee. He is apparently not satisfied with the diagnosis of amyotrophic lateral sclerosis, because of the statement of Oppenheim that this is a disease of middle age, and prefers to reserve the diagnosis pending anatomical researches upon such cases.

JOHN D. COMRIE.

**UNILATERAL CONGENITAL LESION OF MEDULLA AND  
(348) SPINAL CORD; DEATH FROM PONTINE HÆMOR-  
RHAGE.** PURVES STEWART, *Brain*, Spring 1904, p. 89.

In this paper the clinical and post-mortem examination of a case of congenital muscular defect are very carefully recorded. The patient, who was admitted into hospital with cirrhotic Bright's disease, died suddenly from a hæmorrhage into the pons.

There was complete absence of the right sternomastoid, sterno-hyoid, sternothyroid, thyrohyoid, stylohyoid, digastric (posterior belly) and onohyoid muscles. The upper and lower parts of the right trapezius were also absent, the middle part being well developed. The right levator palati and azygos uvulae were smaller than those of the left side. The right middle constrictor of the pharynx was very defective and the styloid muscles were scarcely recognisable. The right and left intrinsic muscles of the tongue were about equal in size. With regard to the larynx, the cricothyroid and posterior crico-arytenoid on the right side were absent, the right lateral crico-arytenoid being represented by a thin film of pale muscle fibres. The transverse and oblique arytenoids were small and the right thyro-arytenoid was feebly developed.

The right half of the larynx as a whole was smaller than the left; especially was this notable in the case of the cricoid.

The right mastoid process was absent and the auditory meatus was narrower on this than on the opposite side.

The right half of the mandible was smaller than the left, and there was a complete absence of all the molar teeth on this side.

"On the back of the neck, from the level of the scalp line to that of the lowest cervical spine, there were two parallel rows of white, flattened, papillomatous projections, congenital, one row on each side of the middle line."

The above description is based on the combined *intra vitam* and post-mortem examinations.

The right hypoglossal and spinal accessory nerves could not be found after death, although very carefully looked for. The right lingual nerve was present and a number of branches passed from it into the hypoglossus.

The right superior and recurrent laryngeal nerves were present, but smaller than those on the left side.

The foramen caecum was abnormally deep.

The right cerebral hemisphere weighed 15 oz., the left 18 oz.

The right cerebellar hemisphere was slightly wider and not so deep as the left; the right side weighed  $2\frac{1}{2}$  oz., the left  $2\frac{1}{2}$  oz.

The right hypoglossal nucleus and adjacent lower part of the accessorio-vago nucleus were absent. The spinal root of the fifth nerve was much smaller on the right side, but this abnormality did not extend down into the cord.

A deep cleft in the position of the antero-median fissure extended from the second to the seventh cervical segments of the cord. The grey matter of the cord in this region was much distorted, and the distortion could be traced up into the medulla. Below the seventh cervical segment the cord showed nothing abnormal. A small number of nerve cells in the right anterior horn were missing in the position of the cleft, but the distortion of

the grey matter rendered it impossible to identify precisely in each segment the particular groups of cells which were absent. The anterior and posterior nerve roots were given off from the grey matter including the cervical region.

On cutting the pons, a number of small hæmorrhages, the largest the size of a pea, were found in its upper third, dorsal to the transverse fibres. The remainder of the pons presented nothing abnormal on microscopic examination.

The author in remarking on the case draws special attention to the presence of a bilateral extensor response in the absence of any interruption of the pyramidal tracts. There was also bilateral, well-marked ankle-clonus.

With regard to the congenital defect, the author has been unable to find any case exactly similar in the literature of nuclear lesions of the medulla or cord.

In this case it appears that there was a separate innervation of the middle third of the trapezius by the cervical anterior roots, the rest of the muscle being supplied by the spinal accessory. The scapula displacement in trapezius paralysis described by Duchenne as the *mouvement de bascule* was perfectly evident, although the acromial bundle of the muscle was well developed. Absence of the sternomastoid was not associated with deformity or defective movement of the head and neck. Notwithstanding the absence of all the infrahyoid muscles there was no impairment of the movements of the hyoid bone.

In the want of marked atrophy of the tongue, with complete absence of the hypoglossal nerve, it seems probable that the one hypoglossal nerve served to supply both sides of the tongue. Thane has shown that there is sometimes a cross loop uniting the two hypoglossal nerves; a less likely view is that the fifth nerve has taken on a vicarious distribution in this case.

EDWIN BRAMWELL.

**A CONTRIBUTION TO OUR KNOWLEDGE OF TUMOURS OF  
(349) THE UPPER CERVICAL CORD AND MEDULLA OBLONGATA.** C. v. RAD, *Deutsch. Ztschr. f. Nervenh.*, Band 26, Heft 3, S. 293.

THE case is described of a man aged 33 years, whose illness commenced by pains in the neck, increasing in severity and frequency; associated was a weakness in the right arm from the shoulder downwards, followed seven months later by a sudden paralysis of the right leg. Nine months after the commencement, the left arm suddenly became affected in a similar manner, and

five months later the left leg. While motor disturbances were more distinctly developed on the right than on the left side, pain and temperature sensibility were diminished on the latter. Epileptoid seizures with dysphagia and dyspnoea occurred as ante-mortem phenomena.

To begin with there was an absence of any symptoms localising the condition of the medulla, and a differential diagnosis of various lesions of the upper cervical cord is given. Potts' disease, pachymeningitis cervicalis hypertrophica, syringomyelia, myelitis and syphilitic disease are in turn excluded. Post-mortem—a tumour (2.5 × 3.5 cm.) was revealed, gliomatous in nature, involving the whole of the medulla and the upper cervical cord, destroying the familiar outlines of the brain stem, and bulging more especially to the right side. A brief description follows of the microscopical findings, although a little more detail was desirable. The right fillet was slightly degenerated, the pyramidal tracts markedly so, though in the right pyramid there remained several bundles of sound fibres. A few fibres in the posterior columns of the cord were affected.

That the tumour would be intra-medullary, not extra-medullary, in location, was indicated, according to the writer, by the rapid development of the double paralysis, the absence of unilateral neuralgia, and the presence of dissociated anæsthesia. The root symptoms (neuralgia and stiffness of head and neck movements) need not always be due to actual involvement of roots; the tumour is frequently situated peripherally and the pains are consecutive to meningeal irritation (Bruns). And, indeed, the actual sensation-conducting paths may be affected in their intramedullary course, leading to mistaken diagnostic localisation (Jolly).

The complete absence of limitation in tongue movements is noteworthy, as also of atrophy in the neck muscles. Probably had the patient lived longer, these would have set in. A widespread furunculosis which supervened not long before death was an indication, according to the writer, of medullary implication.

S. A. KINNIER WILSON.

**A CASE OF JUVENILE APOPLEXY WITH AUTOPSY.** J. (350) RAMSAY HUNT, *Pub. of Cornell Univ. Med. Coll.*, Vol. I., 1904, p. 9.

**A DESCRIPTION of a case of fatal cerebral hæmorrhage in a young man, aged 21.**

The onset was sudden, and attended with deep coma and left hemiplegia. Conjugate deviation of eyes to left. Death in six days.

The autopsy revealed copious hæmorrhage in the right cerebral hemisphere, internal capsule and basal ganglia.

The arteries were thickened, and there were patches of pseudo-senile atheroma. Hypertrophy of the heart, myocarditis and parenchymatous nephritis were present.

The point of great interest in this case is the extraordinary vascular degeneration in so young a man.

AUTHOR'S ABSTRACT.

**ON A CASE OF TUMOUR OF THE CENTRUM OVALE WITH  
(351) CEREBELLAR SYMPTOMS. W. L. ASCHERSON, Sept. 10,  
1904.**

THE object of publishing this case is to emphasise the difficulties which may beset an accurate diagnosis of the site of a cerebral tumour, even in the presence of positive localising signs. The onset of the patient's symptoms was determined by an attack of the nature of influenza, which occurred ten months before her admission into St George's Hospital. During her recovery from this attack the first symptom presented itself in the shape of weakness of the right leg, which was soon followed by rapidly progressive loss of vision, becoming almost absolute in a few months. She began to suffer from severe occipital headache radiating to the vertex, and from vomiting. Ataxia supervened, and by the time she was admitted, she was practically bedridden from inability to walk or stand.

The writer made a diagnosis of tumour of the right lateral lobe of the cerebellum, based upon the following considerations:—

1. Staggering gait, with tendency to fall backwards and to the right.
2. Tenderness in the right occipital region.
3. Early onset and intensity of optic neuritis.
4. Static ataxia of the right arm.
5. Nystagmus, when the eyes were directed to the right.
6. Incompleteness of paralysis with absence of spasm.
7. Variable character of the knee-jerk.
8. Absence of cervical fits.

At the autopsy a large encapsuled tumour of the centrum ovale, underlying the left occipito-parietal region, was found.

The various points upon which the writer's diagnosis was based are next discussed in detail. It is pointed out that although the tumour was of the centrum ovale, the effect of its presence was to cause the brunt of the intra-cranial tension to fall on structures beneath the tentorium, as was clearly shown at the time of the operation. The case is of interest in being one in which a staggering gait was present in the absence of cerebellar disease, and

because it shows that a large tumour may destroy the cortex in the motor area without giving rise to convulsions. Finally, the writer lays stress on the importance of paying most attention, when considering the diagnosis, to the early symptoms which occur before the effects of general intracranial pressure have complicated the clinical aspect of the case.

AUTHOR'S ABSTRACT.

**CEREBRAL SCLEROSIS IN ISOLATED PATCHES ASSOCIATED  
(352) WITH SPECIAL ALTERATIONS OF OTHER ORGANS.**

F. UGOLOTTI, *Riv. di Patol. nerv. e ment.*, Vol. ix., fasc. 8.

THE term "tuberous sclerosis" is applied to a condition in which there is an over-production of neuroglia fibres distributed in circumscribed islands in the cerebral cortex, a disappearance of nerve cells and fibres, and a disorder of the stratification of these last. Frequently, too, there are numerous small nodules in the lateral ventricles. The other parts of the brain are generally normal, the vessels also are normal.

The author, in this paper, gives a clinical, anatomical, and histological account of a case of this kind.

The patient was an imbecile who had suffered from epilepsy since he was seven months old.

At the post-mortem examination numerous nodules, from the size of a grain of maize to that of a nut, were visible to the naked eye in many convolutions of both hemispheres of the brain. Their surface was smooth and glistening. The larger ones were well outlined, and were harder than the others. In the lateral ventricles many small tumours were seen on the surface of the ganglia. Their size was about that of a pea.

The cerebellum, pons, medulla and spinal cord showed nothing microscopically. In the heart muscle there was a small plaque with a yellowish-white colour; in the kidneys there were many nodules of various sizes scattered in the cortex. Lastly, some enormous sebaceous glands were found in the skin of the face.

The histological examination of the tumours of the convolutions showed a diminution of the nerve cells and nerve fibres, and a disorientation of the cells and fibres which were present. With this there was an enormous overgrowth of neuroglia fibres; the number of neuroglia cells was about normal. All these conditions were more marked in the larger and firmer nodules. In the convolutions, which macroscopically appeared normal, nothing unusual was found on examination with the microscope.

The vessels showed no change throughout the brain.

The small tumours in the lateral ventricles consisted of neuroglia fibres. Among these some giant cells were found. These have been considered by some authorities to be nerve cells, in an



embryonal condition, but the author thinks it more probable that they are neuroglia cells.

Microscopical examination did not show anything abnormal in the bulb or cord. The tumours in the kidney were new formations of smooth muscular tissue; the plaque in the myocardium is described as a "myxo-rhabdomyoma."

In conclusion, the author expresses the opinion that the tumours in the brain represent a form of disseminated gliosis. He agrees with Pellizzi and others that there is no sharp line of demarcation to be drawn between hypertrophic sclerosis, neuroglioma, and glioma.

The pathogenesis of "sclerosis tuberosa" is probably to be sought in an error of evolution, which begins in the last months of intrauterine life, and the cause of which is entirely unknown. The absence of any residues of previous morbid processes, the normal condition of the vessels, and the association of changes in several other organs, the kidneys and the heart, suggest a general evolutive disturbance, and practically exclude an inflammatory origin.

R. G. ROWS.

**CONGENITAL WORD BLINDNESS.** SYDNEY STEPHENSON,  
(353) *Lancet*, Sept. 17, 1904, p. 827.

THE author describes two cases of congenital alexia which have come under his observation, and notes 14 cases as described by other writers. Such cases, he thinks, will be found to be commoner than usually supposed, when the existence of such a defect is more generally recognised. The general intelligence in the majority of cases recorded is noted as good, and considerable improvement may take place under careful tuition. Indeed, a case of Nettleship's improved so much as to be able eventually to read quite well, and nineteen years after being first examined the patient had become a lawyer. The diagnosis is not likely to give rise to difficulty unless word-blindness co-exists with a considerable refractive error, as was the case in one of the author's cases. Even in such a case the history should guard one against error, and the fact that the patient has as much difficulty in reading large as small print should suggest the presence of this curious congenital defect.

W. B. DRUMMOND.

**CONTRIBUTION TO THE STUDY OF SENSORY APHASIA.**  
(354) J. DÉJÉRINE and ANDRÉ THOMAS, *Revue Neurolog.* Aug. 15, 1904, p. 805.

A WOMAN, admitted to the Salpêtrière in April 1896, had had an attack of partial right hemiplegia ten months before. Between these dates three Jacksonian attacks had occurred.

On examination, slight general weakness, with no stiffness, was found on the right side; slight analgesia also in the hand and forearm. There was total word-deafness, but in that she heard and distinguished noises and sounds, no psychic deafness. There was no spontaneous speech, and only when much pressed did she reply at all, and then the sounds uttered were not intelligible. There were no repeated utterances. She could not read at all, often holding the paper given to her the wrong way up. Agraphia was total. Intelligence was not materially impaired.

Death occurred in December 1897 from epilepsy.

At the autopsy a large yellow patch of old softening was found on the surface of the left hemisphere, occupying the angular and inferior parietal convolutions, the temporal convolutions not being affected as far as could be seen by the naked eye. Sections stained by the Weigert-Pal method showed that the softening was really much more extensive; the first and second temporal convolutions, among others, were found to be severely affected, and the lesion had extended as deeply as the lateral ventricle. The corpus striatum was not involved.

The main tracts degenerated as a result of this lesion are described. The authors draw particular attention to the impossibility of defining the limits of the lesion before stained sections were made, remarking on the apparent freedom from lesion of the left temporal convolutions in a case of complete word-deafness.

The authors discuss why this patient, with Broca's area intact, should not have jargon-aphasia, concluding that in these cases we must, to some extent, take into account the "mentality of the subject and certain individual psychic variations." They further discuss the question as to whether a lesion of the hinder end of the first temporal convolution can give rise to word-deafness associated with sensory aphasia, and consider that this symptom-complex probably necessitates the involvement of other convolutions as well.

STANLEY BARNES.

#### **ABORTIVE MYXEDEMA, IMPERFECT GROWTH, DIABETES.**

(355) E. APERT, *Nouvelle Icon. de la Salpêtrière*, No. 3, May-June, p. 173.

THIS communication deals with an interesting case of imperfect development in a man of 66, 1 m. 45 in height. He had a rounded face, short neck, well-developed pendant mammae, and a rounded prominent abdomen. The genital organs were rudimentary, the testicles being undescended. The urine amounted to 1½-2 litres per diem and contained 21 grammes of sugar per litre. At 20 years of age his height was only 1 m. 15, and during the next

16 years his height gradually increased by 30 cm., so that at 36 he measured 1 m. 45. The author regards this unusual course of events as being due to a thyroïdal insufficiency in early life which after puberty was less marked, owing to a renewed activity of the thyroid gland, either by regeneration of thyroid tissue or by the compensating action of other glands, as suggested by the enlargement of the mammary and parotid glands. The presence of the glycosuria is also commented upon.

GEORGE MURRAY.

**INFANTILE TYPE OF GIANTISM.** E. BRISSAUD and HENRY (356) MEIGE, *Nouv. Icon. de la Salpêtrière*, No. 3, May-June, p. 165.

THE co-existence of infantilism with dwarfishness has long been noted, as in cretins. This association is, however, not constant, and infantilism may occur, not only in persons of ordinary stature, but also in those who are tall, and there is even an infantile type of giantism. In these giant infants the epiphyses are still un-united, and the power of bony growth apparently persists longer than usual, and so an unusual height is reached. When this excessive tendency to grow persists after the epiphyses are united, the bones grow thicker instead of longer, and giantism is replaced by acromegaly.

In illustration of this infantile type of giantism, a case is recorded of a man, aged 30, who complained of many subjective symptoms. He was 1 m. 85 in height, but had no hair on the face, and very little on the pubes or in the axilla. The testicles were not more developed than those of a boy five or six years of age, and the figure was somewhat feminine in type. The legs were longer in proportion than the arms, as in those who have been castrated. The face showed some of the features of acromegaly, and the feet were large. The case appears thus to be one of an infantile type of giantism which is gradually becoming acromegalic.

GEORGE MURRAY.

**ADIPOSIS DOLOROSA.** J. N. HALL AND C. E. WALBRACH, *Am. (357) Journ. Med. Sc.*, Aug., p. 319.

THE writers describe three cases which showed the four cardinal symptoms of this disease as noted by Vitant, viz., fatty tumour or tumours, pain, asthenia, and psychic disturbances. One of these cases was of the multiple nodular variety, one showed a single painful fatty tumour, and in the third the adipose tissue was diffusely spread over the lower limbs.

In referring to the nature of the disease, they state that as yet in five cases only have post-mortem examinations been held. Of

these, four cases showed changes in the thyroid gland, three showed abnormalities of the pituitary body. From the resemblance of the lesions found to those present in acromegaly, which is also a connective tissue disorder, the writers wish to class the two diseases together.

JOHN D. COMRIE

**ON A SPECIAL FLEXOR REFLEX OF THE TOES.** Remarks (358) on the preceding Communication of W. v. BECHTEREW. KURT MENDEL, *Neurol. Centralbl.*, July 1, 1904, p. 609.

MENDEL and v. BECHTEREW appear to have described independently the same clinical phenomenon, which is as follows:—Percussion is made by means of a rubber hammer on the dorsum of the tarsus or bases of the metatarsal bones. In cases of organic disease of the pyramidal tract, plantar flexion of the toes results, whilst in health or in functional diseases, a dorsal flexion of the toes is observed. V. Bechterew states that this latter is generally, but not constantly, present in health; Mendel says it is a constant phenomenon. The remainder of the papers is taken up by an academic discussion on the question of priority. The fact seems to be that Mendel's observations were made independently of those of v. Bechterew, but some three years later.

PURVES STEWART.

**CONCERNING THE GLUTEAL REFLEX.** V. BECHTEREW, *Neurol.* (359) *Centralbl.*, Sept. 16, 1904, p. 833.

A TAP over the trochanter major is followed by a contraction of the gluteal muscles and a movement of the thigh in the direction of extension.

The erect position is unsuitable, because of the rigidity of the gluteal muscles which commonly accompanies it. The difficulty is to be overcome by slightly flexing the knee. The reflex is often obtainable in health; its presence cannot, therefore, be regarded as pathological. In cases where its activity differs on the two sides of the body, the examination of the reflex may afford diagnostic data of value. It is distinct from the abductor reflex of Schüller.

EDWIN BRAMWELL.

**A STATISTICAL NOTE ON THE SOCIAL CAUSES OF ALCOL-**  
(360) **HOLISM.** W. C. SULLIVAN, *Journ. of Ment. Sc.*, July 1904.

In drawing attention to the social conditions which lead to drunkenness and chronic alcoholism respectively, the author

emphasises the importance of recognising distinct types of drinking. The first is what may be defined as luxury drinking. It is associated with conditions of well-being, and includes various forms of convivial drinking. The second is misery drinking, and stands related to conditions of ill-being. It has its origin in the desire to escape from painful states of all sorts or to overcome the feeling of inability to cope with work. It is associated with unsuitable food, overwork, bad hygiene, etc. Viewed from another standpoint, luxury drinking may be described as convivial, misery as industrial drinking. The two forms may be combined and readily pass into each other. Thus luxury drinking tends to produce conditions which encourage the development of misery drinking. Luxury drinking being more or less intermittent, is not so apt to cause chronic alcoholism as is misery drinking. Now, the more serious of the evils produced by excessive indulgence—racial degeneration, insanity, suicide—are due to chronic alcoholism rather than to drunkenness.

A statistical study of alcoholism in England justifies this view. For several reasons it is difficult to correlate mortality returns for chronic alcoholism with statistics of drunkenness. Accordingly some other index for the prevalence of chronic alcoholism has to be sought. The author has selected for this purpose the returns for suicidal attempts, large areas and long series of years being taken.

A comparison of the various counties in respect of (1) deaths from alcoholism and cirrhosis of liver, (2) arrests for drunkenness, and (3) attempts to commit suicide, show that drunkenness and alcoholism do not coincide in distribution, there being a tendency for the maximum of drunkenness to occur, with the minimum of alcoholism. Confirmation and fresh insight into the subject is obtained when we study returns from composite areas of special industrial character along with comparative mortality rates in occupational groups.

Agricultural and mining districts show respectively the minimum and maximum of drunkenness, and appear relatively equally free from alcoholism. Agricultural districts do not specially favour luxury or misery drinking. Mining districts, with their high wages and generally good home conditions, do not encourage misery drinking; but the population is of low culture and the development of convivial drinking is favoured. In manufacturing towns the conditions of labour and home life make for industrial drinking. There is more female labour here than in agricultural and mining districts, and consequently the women are not so free to attend to rendering home life comfortable. The rate of drunkenness in towns is fairly high, but less than half that in mining districts, while the rate of attempted suicides is

nearly three times that obtaining in mining counties. The mortality figures for alcoholism in large occupational groups, *e.g.* of textile and metal workers, are nearly two or three times as high as in the group of coal miners.

In seaport towns are found the least favourable conditions—work done in spurts of effort, bad housing, low pay, irregularity in work, prolonged hours, freedom from restriction as to drinking during work time, evils attendant on immorality, etc. Yet drunkenness is still below the level it reaches in prosperous mining districts, but chronic alcoholism is enormously developed. The dockers show a death-rate from alcoholism only surpassed by the liquor trade.

The above facts have obvious practical bearings. Primary, convivial drinking is much the more curable of the two forms, frequently subsides as age advances, and is largely influenced by social ideals, etc. Not so with industrial drinking, which tends to get worse with age. When introduced into sober communities it can even weaken the influences that usually tend to control luxury drinking. For checking it we must look to a raised standard of living, and partly to measures restricting drink during work time, and also to hygienic substitutes.

A. HILL BUCHAN.

**THE RELATION OF INEBRIETY TO INSANITY AND ITS  
(361) TREATMENT.** ROBERT JONES, *Lancet*, Aug. 6, 1904.

ALCOHOL is a dys-osmotic, and when ingested it passes with difficulty through the living cellular membrane of the capillaries into the tissues. It induces cell proliferation, deprives the organs of their necessary nutriment, and retards the excretion of waste material, thus giving rise to the view that poisoning from alcohol is an indirect toxæmia arising from the production and accumulation of leucomaines.

The dehydrating action of alcohol upon protoplasm is highly injurious.

The author enumerates the causes of inebriety and remarks that the poor drink in order to get a good meal—mistaking stimulation for nutrition.

*Physiological effects.*—The stages of intoxication are described, as also the pathological effects of alcohol on the tissues.

*Mental effects.*—The mental effects of alcohol differ as to whether they are induced by one large dose as in alcoholic poisoning or in ordinary drunkenness, or whether they are the result of long chronic drinking, even when this is done in "moderation." The mental symptoms of chronic excessive alcoholism may result from long-

continued moderate drinking, more especially if the person is overtaken by a serious illness, or a severe mental shock, also if the drink be spirits.

Persons who become insane and whose symptoms present visual illusions or delusions based upon them, or whose delusions are grandiose, boastful, or relate to sexual perversions, such as marital infidelity, or are suspicious and persecutory in their character, are almost invariably suffering from insanity, directly or indirectly caused by alcohol.

The forms of mental disorder resulting from alcohol are: (a) gross dementia; (b) maniacal excitement due to hallucinations; or (c) permanent delusional states. It is often not the quantity imbibed, but the susceptibility of the brain and nervous system through inherited or other vice which determines whether a given case shall be considered as insane, an inebriate, or a chronic drunkard.

Paramnesia and loss of orientation are characteristic symptoms of alcoholic insanity, and a peculiarity of chronic drinkers is the impulsiveness of all their reactions when excited.

Inebriety is of two kinds: one characterised by intermittence seen in the periodic drinker, and the other in the constant or habitual toper. The former variety generally occurs between twenty and thirty years of age. In women it often occurs coincidentally with the period of menstruation. Periodic drinkers are generally of unstable parentage—alcohol, epilepsy, insanity, or neuralgia. After a bout of drinking the victim either becomes despondent and suicidal, or violent and frenzied from delirium tremens. In the intervals of drinking they probably fulfil their natural obligations to their dependents and to society. In these cases large quantities of alcohol may be imbibed during the drink period without any of the physiological effects, such as drunkenness or motor paralysis, usually seen in the ordinary drunkard.

On the other hand, the constant drinkers are persons usually in later life; they are morally and intellectually depraved. No amount of argument is of any avail in preventing persons in either of these two classes from drinking.

Most of the *criminal insane* are inherently mentally defective. They are suspicious, habitually sullen or unreliably excitable, and vary in their conduct from hour to hour. They are of low intellectual capacity, and are unable to follow a train of reasoning or to behave reasonably in daily life. They are creatures of impulse, and not amenable to method and order.

*Motor symptoms.*—Alcohol induces epilepsy, especially in those who have suffered from head injuries.

*Sensory symptoms.*—Visceral and other hallucinations: complaints as to electricity and machines are quite frequent. The

disturbed sensation may be due to the anæsthetic effect of the alcohol acting peripherally or centrally.

*Susceptibility.*—Alcohol has an affinity for the highest developed functions—both mental and physical—and stress of a particular organ renders that organ more vulnerable. Alcoholic peripheral neuritis has occurred in the right arm of a barmaid, whilst the lower extremities have escaped entirely.

*General results of alcohol.*—The amylic alcohols in crude whisky probably have a more injurious and a different effect from that of the lighter wines. Richet states that the more volatile the spirit, the more marked are the anæsthetic effects of alcohol; the less volatile it is, the more marked are the convulsive effects; and that the more soluble in water the alcohol may be, the more marked are the inebriating effects.

Beer drinking gives rise to malnutrition, as a result of gastric and intestinal irritation; spirit drinking causes glandular cirrhosis.

*Heredity.*—Heredity is as characteristic of alcoholism as it is of any other form of moral insanity, and occurs in about 33 per cent. of all cases of alcoholic insanity. The offspring of the alcoholic are more liable to suffer from delirium tremens and violent mania after taking alcohol, even after a single debauch, than the drunkard without heredity, who, on the other hand, suffers more often from cirrhosis of the liver and kidneys.

The best antidotes to drinking tendencies lie in sanitation and morality, pure water, fresh air, light and sunshine, rational pleasures, education as to the value of food, how to select it and how to cook it. The best auxiliaries to temperance are the influence of education, the impressions given by social usage, increased facilities for physical exercise and recreation, and the controlling factor of public opinion as to the effects of alcohol.

*Treatment.*—With but few exceptions the total and sudden deprivation of alcohol is beneficial. Sleep and nourishment form the keynote of treatment. Sleep is obtained by out-door exercise; failing this, by bromides. Avoid morphine. For motor restlessness give hyoscine ( $\frac{1}{16}$ th to a  $\frac{1}{8}$ th of a grain) hypodermically. Sulphonal and trional (20 grains t.i.d.) are useful, but increase muscular incoördination, and may disturb digestion. When the pulse is weak, and in elderly persons, give paraldehyde in drachm doses. Look out for retention of urine in cases under the influence of sedatives. Administer simple, easily assimilable food, a little at a time, and peptonised if necessary. Attend to the bowels and improve the appetite. Treat the disturbance of the alimentary system on general principles. Look out for bed-sores in bed-ridden cases. No drug can be relied on for the cure of inebriety.

In so far as the treatment of inebriety by restraint is con-



cerned, the law permits of detention in (1) retreats, or (2) reformatories. Patients enter *retreats* voluntarily, and the detention must not be for over two years. Two documents are necessary: (1) a request for reception, signed before a magistrate; and (2) a statutory declaration, signed by two friends, also before a magistrate or a commissioner of oaths. An inebriate may voluntarily enter a retreat without a magistrate's attestation, but in this case he cannot be detained against his will.

Reformatories are two kinds—viz. (1) State reformatories established (*a*) for the more refractory cases in other reformatories, and (*b*) for those committed from the assizes or sessions; and (2) certified reformatories for criminal and police-court cases.

H. DE M. ALEXANDER.

## PSYCHIATRY.

### A CONTRIBUTION TO THE STUDY OF DEMENTIA PRÆCOX.

(362) W. SERBSKY, Moscow, *Ann. Med.-Psych.*, No. 3, 1903, Nos. 1 and 2, 1904.

SERBSKY recognises three types of dementia præcox:—

1. In some cases there is no acute stage, and the onset of the disease cannot be exactly defined. One sees only a slow and progressive enfeeblement of cerebral activity. Gradually the patient becomes abstracted, forgetful, and unfit for work. Sometimes the process is arrested at a moderate dementia which involves, however, the whole sphere of psychical activity. At other times a very marked decline occurs. Weakness of judgment, intellectual insufficiency, obtuseness, indifference to surroundings, absence of all interest and energy are very noticeable. These symptoms are at times seen in early life, and may develop in apparently well endowed and promising individuals. Development is not simply arrested, but is regressive. Though some patients may become able to follow their usual work, involving no unusual effort, anything new they are incapable of.

2. In other cases the disease, evolving on a basis of general psychical enfeeblement, is accompanied, especially at the beginning, by extremely varied symptoms, more or less acute, and even violent in character. There may also be hypochondriacal delusions, a tendency to mysticism, self-accusation, non-systematised delusions of persecution, and grandiose ideas, with or without hallucinations. Sometimes the tendency to argue in a childish manner is dominant, at others magniloquent discussions on the most difficult subjects are carried on. There may be distinct

obsessions, and also impulsiveness. Depression is often present at first, and may exist with attacks of excitement. Obtusion, apathy, indifference are frequently prominent from the commencement. It is worthy of note that a large number of patients give the impression that their madness is feigned, or at least that nothing serious affects them.

Of this type there are three sub-varieties, viz., hebephrenic, katatonic, and paranoid. Most cases, however, show symptoms which make it difficult to attach them to one or another sub-variety.

3. To this type, which most frequently follows acute mental confusion, the author gives the name of secondary progressive dementia. Here the acute period does not show that peculiar shade characteristic of the preceding type, and so the diagnosis is often impossible until the end. Improvements and relapses take place, with a general and progressive decline of mental activity. The terminal condition is often identical with that of typical hebephrenic dementia.

Two essential features in the *diagnosis* are the onset during adolescence, and the rapid production of a more or less pronounced incurable dementia. No constant physical signs have been discovered. The diagnosis of the first type is easy. In neurasthenia and overwork the entire field of psychical activity is not affected.

Typical hebephrenic, katatonic, and paranoid dementia, and cases where the dementia is marked from the outset, are not difficult to distinguish.

Long observation is, however, necessary for the diagnosis between it and amentia, the periodical psychoses, and attacks of delusion among the degenerate. Secondary dementia is excluded by the age of onset and the peculiar character of the acute stage. It must be admitted, however, that during adolescence there may develop other disorders passing into dementia, which as yet are indistinguishable from dementia *præcox*.

The morbid anatomy is unknown.

The theory of auto-intoxication (particularly by sexual products) is considered by the author to have no solid basis. He is of opinion that there is not only degeneration, but also an original antenatal weakness of constitution which the demands made upon the organism at puberty bring to light.

With regard to *treatment*, serumtherapy and organotherapy may in the future become of service, but at present systematic and appropriate work and exercise are our only means of checking the progress of the disease. Prolonged confinement in bed, except during periods of aggravation or excitement, is injurious.

W. T. JAMES.

**THE STUDY OF A NON-DEMENTING PSYCHOSIS BY LABORATORY METHODS.** CHARLES L. DANA, *Pub. of Cornell Univ. Med. Coll.*, Vol. i., 1904.

THE mental condition of a case of chronic insanity, associated with a certain degree of dementia, is submitted for study. At three intervals, covering a period of three years, his mental state was studied by Professor Cattell, assistant to the writer, by means of all the various psychological laboratory methods. It was shown by the investigation that the dementia had become completely stationary. Hence the conclusion: degenerative insanity may, during its course, come to a complete standstill.

AUTHOR'S ABSTRACT.

---

## Reviews

**OÉCITÉ ET TABES (ÉTUDE CLINIQUE).** ANDRÉ LERI. Pp. viii. + 246. Paris: Rueff. 1904.

No morbid conditions have within recent years been more provocative of discussion than tabes, tabo-paralysis, and general paralysis. It is scarcely paradoxical to maintain that maybe the very wealth of material forthcoming has not been without effect in giving rise to the many conflicting theories which face us in regard to one or all of them. It has seemed, perhaps, sometimes, that there has been over-elaboration of clinical detail at the expense of the pathological, or *vice versa*; hence a contribution to the subject, or to any part of the subject, is to be welcomed which avowedly deals with both sides in a full, though it may not be exhaustive, manner.

The thesis of M. André Leri on the relation between tabes and optic atrophy is divided into two parts, the first of which—a purely clinical study of forty-five cases of associated tabes and blindness—has just been published. We are promised the complementary anatomical results in a second volume.

Here, at any rate, we have sufficient clinical material to substantiate or demolish current hypotheses on the relationship of tabes and amaurosis—material the value of which is enhanced when it is stated that, owing to circumstances which need not be mentioned here, Leri has had at his disposal cases which have been carefully followed, sometimes through many years, some of which

were described in Martin's thesis (Berne) as long ago as 1890. Exceptional opportunity has thus been afforded of watching the advance of pathological processes through long years, and thus of corroborating or correcting theories elaborated perhaps without sufficient basis.

Granting that, say, 25 per cent. of all tabetics become amaurotic eventually, or rather may have an associated amaurosis, do we believe that the latter is but a grave complication of a disease already grave enough in itself, or do we agree with Benedikt (1881) and with Fournier (1882) in denominating this amaurosis as a *fruste* form of tabes, altogether benign in its significance, in fact able somehow or other to exert a retarding influence on the spinal manifestations of the disease? Déjérine and Martin (1889) did not hesitate to say that optic atrophy supervening at the onset of tabes practically always arrests the evolution of the sclerosis in the posterior columns, diminishing at the same time the lightning pains and other sensory phenomena. Pierre Marie, however (1892), while admitting that cases of tabes in which the optic nerves are affected ought to be classed as superior or cerebral tabes, other organs remaining almost unaffected, could not allow any direct action of the optic neuritis on the disease.

It must be evident that there is something "special" in the association of tabes and optic atrophy. The feeble development of spinal symptoms cannot surely be explained as a mere coincidence, it is of such frequent occurrence. Yet no more can it be explained by a mysterious influence exercised by the optic atrophy on the evolution of the morbid process elsewhere.

To aid in the solution the author propounds two questions which his thesis is an attempt to answer:—

1. What are the clinical characters of the amaurosis which is accompanied by tabetic phenomena?

2. What are the clinical relations between these phenomena and the amaurosis?

The whole ground is covered with admirable thoroughness.

The troubles of vision have no characteristic feature (subjective or objective) which would distinguish them unfailingly from similar conditions which one may meet in other syphilitic affections of the nervous system—general paralysis, for instance.

Next, all the ordinary clinical manifestations of tabes are classified into cerebral and spinal, and methodically analysed and compared in the two groups under consideration. It is just here that the work is of the greatest value, and the importance of following cases through many years amply evidenced. Even though optic atrophy be the initial symptom, it does not follow (*pace* Benedikt) that the patient will remain preataxic until the end: abundant clinical observation, concisely classified by the author,

shows that tabes of the most pronounced type, with incoördination, cachexia, and all the familiar signs, may develop eventually. This is not, of course, a new observation; but the wealth of detail adduced by Leri in support of the contention is irresistible.

It is shown that tabetics with optic atrophy present a symptom-complex of cephalic or cerebral phenomena exactly akin (apart from the amaurosis) to that found in ordinary tabes, whereas the spinal manifestations are more slight, usually, than in the latter, but there is no clinical relation between the two classes or groups of symptoms. The advent of optic atrophy has no beneficial action on the lightning pains or ataxia, be they of recent or of old development. Clinically tabetic amaurosis and ordinary tabes are sometimes combined; they are most frequently independent; their etiology is the same: they are but two separated localisations of the same morbid process; and as so often the medullary lesions in the former are absolutely minimal, the mental so predominant, it is almost an abuse of language to denominate *tabetic* amaurosis what is really *sypilitic* amaurosis. In short, the author would prefer the use of the word *tabétisant* (Marie and Switalski), employed to indicate that type of tabetic (blind or not) who presents so few of the cardinal signs of the disease, who is often in excellent health apparently, who may never go beyond the preataxic stage, though he *may* become a real tabetic. Here one finds the majority of cases of optic atrophy; they are *tabétisants*, not tabetics. Thus the author is led inevitably from his clinical research to the wider generalisation, that general paralysis, tabes dorsalis and the amaurosis of *tabétisants* are clinically but three localisations of the same pathological process.

S. A. KINNIER WILSON.

**HYSTERICAL MENTAL AFFECTIONS.** By Dr E. RAIMANN.  
Vienna: F. Deuticke, 1904, pp. 395, M. 9.

THIS monograph is based on the results of its author's experience in private practice, in a private sanatorium, in a public asylum, and in the psychiatric clinic of a large public hospital. It thus embraces a study of all grades of society, and in this important respect a broad groundwork has been secured on which to build conclusions. In the preface these are stated in a general way. Hysteria is a psychosis, it is always accompanied by psychical symptoms, and forms a connecting link between neurology and psychiatry. An attempt is made to unify the symptoms and causes, to bridge the gaps, to clear up and eliminate contradictions. In giving details of his cases, which it may here be said are full and exact, Raimann has endeavoured to present the ordinary symptoms

of hysteria, and to avoid rare though interesting cases of an exceptional kind. The latter may be valuable to the specialist, but to the everyday worker it is more important to have presented to him a picture of the disease which he will probably meet, and be able to recognise it in his daily practice.

The introductory chapter is devoted to a definition and general description of the disease. Owing to the great variety of its symptoms the end members of a graduated series of cases present many contrasts and even apparent contradictions, but in all certain psychical alterations are present. These are not always prominent, but may take a quite subordinate position and require search to discover. The cardinal symptom is considered by the author to lie in the patient's abnormal suggestibility, in his morbid reactivity to external influences.

The second chapter deals with what is called the hysterical character. In all hysterics certain psychical attributes may be observed, which bring about an abnormal sensitiveness of reaction to the environment. These in their sum total constitute the hysterical character. In this suggestibility is a most prominent feature. Another is the increased attention which is turned to the state of the feelings and to the whole Ego ("das liebe Ich"). There is a morbid hypochondria which enters into all departments of mental life and not only that of bodily sensation. From this the steps are easy to an unconscious or half-conscious simulation, which is another prominent feature. Irritability with weakness is also observable in the nervous mechanism. The hysterical character is not a matter of temperament. All four may manifest it, the sanguine and the lymphatic equally with the bilious and melancholic. The perverted egoism referred to above accounts for many prominent symptoms, especially for the characteristic sensory and emotional disturbances, for the very great prevalence of the sexual element, and for the weakness of volition.

The third chapter is devoted to the clinical examination of hysteria, and the classification of its various forms resulting therefrom. Dr Raimann considers that, though the symptoms are so various, there is no insuperable difficulty in the way of a simple classification. The hysterical fit is regarded as the classical symptom of the disease, and the various forms of hysteria which he recognises are grouped round this. He distinguishes five varieties; those associated with the hysterical seizure, those accompanied by delirium, and by confusion, other acute hysterical psychoses resembling ordinary mental disease, and lastly chronic forms of hysterical psychosis. Very full descriptions of illustrative cases are given under each of these headings. It will be observed that the fourth variety is not very clearly defined, and in consequence the tendency arises to place all cases not clearly belong-

ing to other groups in this one, thus sacrificing accuracy for the sake of apparent simplicity.

In the fourth chapter the question of etiology is considered. The causes are recognised as predisposing and exciting. As regards age, it commonly makes its first appearance in adolescence, but it may be met with at all ages from 2 to 74. The influence of sex, race, condition as to marriage, and occupation is touched on. The most fertile predisposing cause is recognised to be heredity, but no new facts or theories are advanced under this heading. Chronic alcoholism is referred to as a predisposing factor in some. Of exciting causes, acute alcoholism is first referred to, as well as other intoxications. The author does not consider that disease of the sexual organs has any special relation to hysteria, though he has devoted careful investigation to this point. Neither does he regard traumatism as at all a frequent cause. He is inclined to think that in almost all cases the cause is a psychical one, such as sudden and intense emotional disturbance.

The fifth chapter is mainly devoted to a short historical review of the various suggestions which have been made as to what the essence of hysteria is. The author himself is inclined to the view that hysteria is a disease of psychical origin, abnormal suggestibility being the chief morbid condition. One looks in vain, however, for some light on the explanation of this diseased state.

The sixth and seventh chapters are devoted to differential diagnosis and prognosis. In the former no attempt is made to distinguish it from organic nervous disease. This is probably a mistake, as it is well recognised that organic disease of the nervous system may be associated with hysteria, and may be overlooked if too much attention is devoted to the latter, which is probably the more prominent but less important symptom. As to prognosis, the disease is looked on as being in the majority of cases eminently curable, but liable to relapse. The climacteric often sees it finally depart.

Treatment is discussed in the eighth chapter. Prophylaxis is important, and directions are given with this end in view. As the disease is psychical the treatment is on the same lines. Naturally, much is said of hypnotism. The author's experience is, on the whole, not favourable to it. Much more good results from the repeated impression of suggestions during normal consciousness, and it is in this that the personality of the physician counts for much. Ovariectomy is strongly condemned, and the statistics of cases he refers to seem to prove this without doubt.

In the last chapter the various ways in which hysterical patients may come into the law courts are adverted to. Whether as witnesses, as plaintiffs, or as indicted criminals, the greatest

circumspection must be used. Each case requires minute and careful investigation.

The book closes with a very full and valuable list of references to the subject.

On the whole, the book is a useful one, as summarising the facts about hysteria and its relations to mental disease, but one misses the suggestiveness and brightness of several French works on this matter, notably Janet's, and it is not possible to say that it throws much new light on an exceedingly interesting but difficult subject.

JAS. MIDDLEMASS.



# Bibliography

## ANATOMY

- EDINGER. Vorlesungen über den Bau der nervösen Zentralorgane des Menschen und der Tiere. Bd. ii., F. C. W. Vogel, Leipzig, 1904, M. 5.
- VAN BAMBEKE. Rapport sur le mémoire de M. Joris intitulé: Histogenèse du neurone. *Bull. de l'Acad. Roy. de Méd.*, T. xviii., No. 6, 1904, p. 315.
- AZOULAY. Les neuro-fibrilles d'après la méthode et les travaux de S. Ramon y Cajal. *Presse méd.*, août 24 et sept. 14, 1904.
- MARINESCO. Nouvelles recherches sur les neuro-fibrilles. *Rev. Neurol.*, août 15, 1904, p. 813.
- F. NISSL. Arbeiten, histologische und histopathologische über die Grosshirnrinde. G. Fischer, Jena, M. 40.
- GURWITSCH. Morphologie und Biologie der Zelle. G. Fischer, Jena, 1904, M. 9.
- WIEDERSHEIM. Bemerkungen zur Anatomie des menschlichen Ammonshorns. *Monatsschr. f. Psychiat. u. Neurol.*, Bd. xvi., H. 2, 1904, S. 283.
- LEWANDOWSKY. Untersuchungen über die Leitungsbahnen des Truncus cerebri und ihren Zusammenhang mit denen der Medulla spinalis und des Cortex cerebri. Gustav Fischer, Jena, 1904.
- V. MONAKOW. Die Stabkranzfasern des unteren Scheitellappchens und die sagittalen Strahlungen des Occipitallappens. *Monatsschr. f. Psychiat. u. Neurol.*, Bd. xvi., H. 2, 1904, S. 287.
- GUISEPPE PANEGROSSI. Weiterer Beitrag zum Studium der Augenmuskelnervenkerne. *Monatsschr. f. Psychiat. u. Neurol.*, Bd. xvi., H. 2, 1904, S. 268.

## COMPARATIVE ANATOMY—

- KRAUSE und KLEMPNER. Untersuchungen über den Bau des Centralnervensystems der Affen. G. Reimer, Berlin, 1904, M. 4.50.
- O. CHARNOCK BRADLEY. Neuromeres of the Rhombencephalon of the Pig. *Rev. Neurol. and Psychiat.*, Sept. 1904, p. 625.
- GOUREVITCH. On the External Appearance of the Nervous Elements of the Cerebellar Cortex of Young Mammalia. *Journ. Ment. Pathol.*, Vol. v., Nos. 4-5, 1904, p. 118.
- BRODMANN. Demonstrationen aus Cytoarchitektonik der Grosshirnrinde mit besonderer Berücksichtigung der histologischen Lokalisation bei einigen Säugetieren. *Allg. Ztschr. f. Psychiat.*, Bd. lxi., H. 5, 1904, S. 765.
- CAVALIÉ. Les ramifications nerveuses dans l'organe électrique de la torpille. *Bibliogr. Anat.*, T. xiii., f. 4, 1904, p. 214.

## METHODS—

- B. POLLACK. Färbungsmethoden. *Monatsschr. f. Psychiat. u. Neurol.*, Bd. xvi., H. 2, 1904, S. 282.
- DE NABIAS. Nouvelle méthode de coloration rapide du système nerveux au chlorure d'or. *Bibliogr. Anat.*, T. xiii., f. 4, 1904, p. 221.
- F. REICH. Ein Apparat zur Bestimmung des Gehirnvolumens: Cerebrovoluminimeter. *Neurol. Centralbl.*, Sept. 16, 1904, S. 839.

## PHYSIOLOGY

- LANGLEY and ANDERSON. On Autogenetic Regeneration in the Nerves of the Limbs. *Journ. Physiol.*, Aug. 22, 1904, p. 418.
- LANGLEY and ANDERSON. The Union of Different Kinds of Nerve Fibres. *Journ. Physiol.*, Aug. 22, 1904, p. 365.

- STARCK. Experimentelles über motorische Vagusfunktion. *Mösch. med. Wchnschr.*, Aug. 23, 1904, S. 1512.
- SWIRSKI. Ueber die Beeinflussung des Vagus-centrums durch das Coffein. *Arch. f. d. gesam. Physiol.*, Bd. civ., H. 5 u. 6, 1904, S. 260.
- GRÜTZNER. Zustandekommen natürlicher Muskelbewegungen. *Monatschr. f. Psychiat. u. Neurol.*, Bd. xvi., H. 2, 1904, S. 284.
- WEYGANDT. Ueber den Einfluss von Hunger und Schlaflosigkeit auf die Hirnrinde. *Monatschr. f. Psychiat. u. Neurol.*, Bd. xvi., H. 2, 1904, S. 288.
- GRABOWER. Die Verteilung und Zahl der Nervenfasern in der Kehlkopfmuskeln und die Hinfälligkeit des Erweiterers der Stimmritze. *Arch. f. Laryngol. u. Rhinol.*, Bd. xvi., H. 2, 1904, S. 189.
- T. BRAILSFORD ROBERTSON. On the "Sham-death" Reflex in Spiders. *Journ. Physiol.*, Aug. 22, 1904, p. 410.

## PSYCHOLOGY

- S. DE SANCTIS. Le problème de la conscience dans la psychologie. *Arch. de Psychol.*, T. iii., No. 12, 1904, p. 379.
- A. LEMAITRE. Observations sur le langage intérieur des enfants. *Arch. de Psychol.*, août 1904, p. 1.
- L. SCHNYDER. L'examen de la suggestibilité chez les nerveux. *Arch. de Psychol.*, août 1904, p. 44.
- J. REINKE. Néovitalisme et finalité en biologie. *Arch. de Psychol.*, T. iii., No. 12, 1904, p. 375.
- PARANT. Dissociation des symptômes angoisse et anxiété. Marqués et Cie. Toulouse, 1904.
- DEL GRECO. La psiche del simulatore. *Manicomio*, Anno xx., No. 2, 1904, p. 179.
- TH. FLOURNOY. Note sur un songe prophétique réalisé. *Arch. de Psychol.*, août 1904, p. 58.
- ROSSI. Della memoria e dell' immaginazione sociale. *Manicomio*, Anno xx., No. 2, 1904, p. 207.
- TH. FLOURNOY. Chorégraphie somnambulique. *Arch. de Psychol.*, T. iii., No. 12, 1904, p. 357.
- CHATELAIN. Testament d'un simple d'esprit. *Ann. méd.-psychol.*, Sept.-Oct. 1904, p. 221.
- MEUNIER. Remarks on a Case of Precocious Attention to Esthetic Sensations. *Journ. Ment. Pathol.*, Vol. v., Nos. 4-5, 1904, p. 136.
- A. PICTET. Observations sur le sommeil chez les insectes. *Arch. de Psychol.*, T. iii., No. 12, 1904, p. 337.

## PATHOLOGY

- WESTPHAL. Demonstration mikroskopischer Präparate eines seltenen Falles von Missbildung des Rückenmarkes. *Allg. Ztschr. f. Psychiat.*, Bd. lxi., H. 5, 1904, S. 765.
- A. S. JOSKE. Two Cases of Macrocephaly. *Australian Med. Gaz.*, Vol. xxxii. No. 7, 1904, p. 342.
- A. RYDEL. Sur l'anatomie pathologique d'une forme d'hérédito-ataxie cérébelleuse. *Nouv. Icon. de la Salpêtrière*, T. xvii., No. 4, 1904, p. 289.
- A. LÉRI. Étude de la rétine dans l'amaurose tabétique. *Nouv. Icon. de la Salpêtrière*, T. xvii., No. 4, 1904, p. 304.
- RABAUD et ANTHONY. Étude anatomique et considérations morphogéniques sur un exencéphalien proencéphale. *Bibliogr. Anat.*, T. xiii., f. 4, 1904, p. 183.
- J. RAMSAY HUNT. The Retrograde Atrophy of the Pyramidal Tracts. *Journ. Nerv. and Ment. Dis.*, Aug. 1904, p. 504.
- RIEDER. Contribution à l'étude des kystes parasitaires du cerveau causés par le cystique du ténia échinocoqus. *Thèse*. F. Alcan, Paris, 1904.
- SATO. Über Cysticerken im Gehirn des Menschen. *Deutsche Ztschr. f. Nervenheilk.*, Bd. xxvii., H. 1 u. 2, 1904, S. 24.
- BRISAUD et BAUER. A propos des modifications de la moelle consécutives aux amputations de membres chez le tétard. *Rev. Neurol.*, sept. 15, 1904, p. 929.
- GEORGE LAMB and WALTER K. HUNTER. On the Action of Venoms of Different Snakes on the Nervous System. *Lancet*, Aug. 20, 1904, p. 518.

- W. D'ESTE EMERY. A New Pathogenic Bacterium causing Basal Meningitis in Infants. *Lancet*, Aug. 27, 1904, p. 593.  
 WEYGANDT. Verhalten des Gehirns bei Situs viscerum transversus. *Allg. Ztschr. f. Psychiat.*, Bd. lxi., H. 5, 1904, S. 762.  
 JULIUS GRINKER. Tuberculosis of the Nervous System. *Chicago Med. Recorder*, July 1904.  
 KOCH. Ueber eine seltene Form der Tuberkulose des Kleinhirns. *A. Hirschwald*, Berlin, 1904, M.—80

## CLINICAL NEUROLOGY AND PSYCHIATRY

### GENERAL—

- XIVe. Congrès des Médecins Aliénistes et Neurologistes de France et des Pays de Langue Française, Pau, 1-8 août, 1904. *Rev. Neurol.*, août 31, 1904.  
 LEYDEN and GOLDSCHIEDER. Die Erkrankungen des Rückenmarkes und der Medulla oblongata. II. A. Hölzler, Wien, 1904, M. 17.20.  
 FRITZ SCHIFFER. Bericht über 1150 Fälle von Nervenkrankheiten im Kindesalter. *Jahrb. f. Kinderheilk.*, Aug. 15, 1904, S. 388.  
 SIEMERLING. Wert und Bedeutung der Cytodiagnose für Geistes und Nervenkrankheiten. *Allg. Ztschr. f. Psychiat.*, Bd. lxi., H. 5, 1904, S. 758.  
 E. HAHNLE. Der heutige Stand der Erblichkeitsfrage in der Neuro- und Psychopathologie. *Neurol. Centralbl.*, Sept. 16, 1904, S. 843.  
 L. PIERCE CLARK. Notes on Some Uncommon Forms of Nervous Diseases. *Med. Rec.*, July 23, 1904.  
 NEURATH. Die nervösen Komplikationen und Nachkrankheiten des Keuchhustens. F. Deuticke, Wien, 1904, M. 8.  
 BARDET. De la prépondérance des lésions du système cérébro-spinal dans l'étiologie et le pronostic de la mort apparente. *Thèse de Paris*, 1904.

### MUSCLES—

- L. SPILLMANN. Atrophie musculaire myopathique (type juvénile d'Erb) et nerfs vasculaires. *Rev. méd. de l'Est*, juillet 15, 1904.  
 FR. JAMIN. Experimentelle Untersuchungen zur Lehre von der Atrophie gelähmter Muskeln. G. Fischer, Jena, 1904.  
 ALBERT KNAPP. Ein Fall von doppelseitigem Schwund der Wadenmuskulatur. *Monatsschr. f. Psychiat. u. Neurol.*, Bd. xvi., Ergänzungsheft, Aug. 1904, S. 161.

### PERIPHERAL NERVES—

- G. ALLAIRE. Paralyse par compression des nerfs cubital et médian. *Ann. d'Électrobiol. et de Radiol.*, Vol. vii., No. 2, 1904, p. 188.  
 P. BLACKALL. Epidemic Polyneuritis. *Austral. Med. Gaz.*, Vol. xxiii., No. 7, 1904, p. 347.  
 MEDÉA and GEMELLI. A Case of Toxic Polyneuritis probably due to Aniline Poisoning. *Journ. Ment. Pathol.*, Vol. v., Nos. 4-5, 1904, p. 113.  
 EARLE PAGE. A Case of Acute Ascending Paralysis. *Austral. Med. Gaz.*, Vol. xxiii., No. 7, 1904, p. 348.  
 MOSSE. Report on an Outbreak of Beri-beri amongst Prisoners of War at St Helena. *Journ. of the R.A.M.C.*, Sept. 1904, p. 237.

### SPINAL CORD—

- Tabes.**—DETERMANN. Die Diagnose und die Allgemeinbehandlung der Frühstadien der Tabes dorsalis. C. Marhold, Halle, 1904, M. 2.50.  
 KURT FRANK. Wirbelerkrankung bei Tabes dorsalis. *Wien. klin. Wchnschr.*, Aug. 25, 1904, S. 919.  
 IDELSOHN. Ein Beitrag zur Pathologie und Histologie des "tabischen Fusses." *Deutsche Ztschr. f. Nervenheilk.*, Bd. xxvii., H. 1 u. 2, 1904, S. 121.  
 CARL WEIGERT. Bemerkung über eine Kleinhirnveränderung bei Tabes dorsalis. *Neurol. Centralbl.*, Aug. 16, 1904, S. 738.  
**Friedreich's Ataxia.**—GUENOT. Contribution à l'étude clinique, anatomopathologique et étiologique de la maladie de Friedreich. Rey et Cie, Lyon, 1904, 2 fr. 50.

- Family Spastic Paraplegia.**—NEWMARK. Über die familiäre spastische Paraplegie. *Deutsche Ztschr. f. Nervenheilk.*, Bd. xxvii, H. 1 u. 2, 1904, S. 1.
- Pressure Paraplegia.**—EPS. Klinische und pathologisch-anatomische Untersuchung eines Falles von Wirbelcaries und Compressionsmyelitis auf tuberkulöser Basis mit besonderem Berücksicht der sekundären Degenerationen. F. Fues, Tübingen, 1904, M. 1.20.
- ALFRED GORDON. A Microscopical Study of the Spinal Cord, not Compressed by Displaced Vertebrae, in a Case of Pott's Disease. *Journ. Nerv. and Ment. Dis.*, Aug. 1904, p. 528.
- Poliomyelitis Anterior Acuta.**—R. B. WADE and W. T. LITCHFIELD. The Recent Epidemic of Infantile Paralysis. *Austral. Med. Gaz.*, Vol. xliii, No. 7, 1904, p. 344.
- Arteriosclerosis.**—JOSEPH COLLINS and EDWIN ZABRISKIE. Arteriosclerosis of the Spinal Cord. *Med. Rec.*, Sept. 1904, p. 361.
- Disseminated Sclerosis.**—FRED. S. PALMER. Early Manifestations of Insular Sclerosis, with a Table showing the Modes of Onset in Fifty Cases, taken from Notes made at the National Hospital in Queen Square. *Med. Press*, Sept. 7, 1904, p. 243.
- Syringomyelia.**—ARTHUR ROSENFELD. Über traumatische Syringomyelie und Tabes. *Samml. Klin. Vortr.*, Aug. 1904, S. 247.
- Hæmatomyelia.**—TANIGUCHI. Ein Fall von Hæmatomyelie im Anschluss an eine Carcinom-metastase im Lendenmark. *Deutsche Ztschr. f. Nervenheilk.*, Bd. xxvii, H. 1 u. 2, 1904, S. 148.
- DINKLER. Beitrag zur Symptomatologie und Anatomie der Apoplexia spinalis. *Monatschr. f. Psychiat. u. Neurol.*, Bd. xvi, H. 2, 1904, S. 285.
- Tumour.**—J. M. GILL. Glioma of the Spinal Cord. *Australian Med. Gaz.*, Vol. xliii, No. 7, 1904, p. 339.
- Spina Bifida.**—J. F. MATTEI. Un cas de spina bifida cervical, avec examen histologique. *Thèse*. Firmin, Montane et Sicardi, Montpellier, 1904.
- E. W. TAYLOR. Sacral Spina bifida with reference to the Skin Representation of Sacral Nerve Roots. *Boston Med. and Surg. Journ.*, Aug. 25, 1904, p. 208.
- Concussion.**—STOLPER. Zur Lehre von der Rückenmarkserschütterung. Erwiderung an Herrn Dr Schäffer. *Vierteljahrsschr. f. gericht. Med.*, Juli 1904, S. 67.
- HUGO STARK. Ueber Vorderhornkrankung nach Trauma. *Monatschr. f. Psychiat. u. Neurol.*, Bd. xvi, H. 2, 1904, S. 291.

#### BRAIN AND MEMBRANES—

- UGOLOTTI. Sclerosi cerebrali tuberose associate a speciali alterazioni di altri organi. *Riv. di Patol. nerv. e ment.*, Vol. ix, f. 8, 1904, p. 361.
- GROBER. Diabetes insipidus mit cerebralen Herdsymptomen. *Münch. med. Wochenschr.*, Sept. 13, 1904, S. 1633.
- HENRY S. WIEDER. Brain Injury. 1. Loss of One Quarter of Brain Substance with Consciousness and Ability to Talk retained until Death. 2. Fracture of the Skull with Porencephalia. *Am. Med.*, Phila., July 23, 1904.
- Meningitis.**—W. G. BARRAS. Meningeal Infection by the Diplococcus Pneumoniae, simulating Infective Cerebro-spinal Meningitis. *Lancet*, Aug. 27, 1904, p. 590.
- BRAUN. Pachymeningitis externa fungosa. *Vierteljahrsschr. f. gericht. Med.*, Juli 1904, S. 71.
- RICHTER. Gangränöses Pachymeningitis und Wasserstoffsuperoxyd Merg zum Blutnachweis. *Monatschr. f. Ohrenheilk.*, Bd. xxxviii, No. 7, 1904, S. 300.
- CASSAB. Variations de la formule cytologique au cours des méningites tuberculeuses. *Thèse*. Storck et Cie., Paris, 1904.
- L'HÔTE. Des rémissions prolongées dans la méningite tuberculeuse. *Thèse*. Le Bigot frères, Lille, 1904.

- Hydrocephalus.**—H. LOWENBERG. Treatment of Chronic Internal Hydrocephalus by Lumbar Puncture. *Am. Med.*, Aug. 6, 1904.  
 ALFRED S. TAYLOR. The Treatment of Chronic Internal Hydrocephalus by Autodrainage. *Am. Journ. Med. Sc.*, Aug. 1904.
- Hemiplegia.**—LIEPMANN. Ueber Dissociation der oberflächlichen und tiefen Schmerzempfindung bei cerebraler Hemiplegie. *Neurol. Centralbl.*, Aug. 16, 1904, S. 740.
- Encephalitis.**—SPIELMEYER. Ueber eine epileptische Form des Grosshirnen-  
 cephalitis. *Monatsschr. f. Psychiat. u. Neurol.*, Bd. xvi., H. 2, 1904. S. 291.
- Tumour.**—R. T. WILLIAMSON. The Diagnostic Value of Hemiplegia of Gradual Onset as a Sign of Cerebral Tumour. *Practitioner*, Sept. 1904, p. 321.  
 MOINDROT. De la ponction lombaire dans les tumeurs cérébrales. *Thèse*. Storck et Cie, Paris, 1904.  
 W. L. ASCHERSON. On a Case of Tumour of the Centrum Ovale with Cerebellar Symptoms. *Lancet*, Sept. 10, 1904, p. 756.  
 REICH. Hard im Thalamus opticus. *Centralbl. f. Nervenheilk. u. Psychiat.*, Sept. 15, 1904, S. 577.  
 R. T. WILLIAMSON. A Case of Multiple (Tuberculous) Cerebral Tumours. *Med. Chron.*, Sept. 1904.  
 J. GRAY CLEGG. Epibulbar Melanotic Sarcoma. *Lancet*, Sept. 10, 1904, p. 750.  
 H. J. WHITACRE. A Case of Tumor of the Brain: Operation: Recovery. *Cincinnati Lancet-Clinic*, Aug. 6, 1904.
- Abscess.**—ALICE E. WAKEFIELD. Report of a Fatal Case of Latent Temporo-Sphenoidal Abscess of Otitic Origin, followed by Multiple Secondary Cerebral Abscesses. *Arch. of Otol.*, Vol. xxxiii., No. 4, 1904, p. 273.  
 GORHAM BACON. Report of a Fatal Case of Brain Abscess of Otitic Origin. *Arch. of Otol.*, Vol. xxxiii., No. 4, 1904, p. 269.  
 WELLS P. EAGLETON. Remarks on a Double Cerebellar Abscess; Operations and Recovery. *Arch. of Otol.*, Vol. xxxiii., No. 4, 1904, p. 283.  
 ALT. Zwei gheilte Fälle von Kleinhirnsabscess. *Monatsschr. f. Ohrenheilk.*, Bd. xxxviii., No. 7, 1904, S. 297.  
 GERBER. Rhinogener Hirnabscess. *Arch. f. Laryngol. u. Rhinol.*, Bd. xvi., H. 2, 1904, S. 208.  
 EULENSTEIN. Bemerkenswerter Fall von multiplen intrakraniellen Komplikationen bei chronischer Mittelohreiterung. *Ztschr. f. Ohrenheilk.*, Bd. xlvii., 1904, S. 84.  
 SIGNARD. Contribution à l'étude de la thrombo-phlébite des sinus caverneux d'origine otitique. *Thèse*. Joannin et Cie., Paris, 1904.
- General Paralysis.**—RAECKE. L'Étiologie de la paralysie progressive. Poinet, Lyon, 1904.  
 ABRAHAM und ZIEGENHAGEN. Über cytodagnostische Untersuchungen bei Dementia paralytica. *Allg. Ztschr. f. Psychiat.*, Bd. lxi., H. 5, 1904, S. 775.  
 TOMASINI. Su alcune lesioni trofiche (ulcerazioni intestinali) nella paralisi progressiva e loro importanza medico-legale. *Manicomio*, Anno xx., No. 2, 1904, p. 277.

## GENERAL AND FUNCTIONAL DISEASES—

- Chorea.**—W. L. JOHNSON. Report of a Case of Senile Chorea. *St Louis Courier of Med.*, July 1904.  
 DE BUCK. Un cas de chorée chronique progressive avec autopsie. *Journ. de Neurol.*, Sept. 5, 1904, p. 321.
- Epilepsy.**—HENRY J. RHETT. Epilepsy. *Washington Med. Ann.*, Sept. 1904.  
 DONATH. Die Bedeutung des Cholins in der Epilepsie. Nebst Beiträgen zur Wirkung des Cholins und Neurins sowie zur Chemie der Cerebrospinalflüssigkeit. *Deutsche Ztschr. f. Nervenheilk.*, Bd. xxvii., H. 1 u. 2, 1904, S. 71.  
 ETIEVANT. Epilepsie d'origine nasale. Coulet et Poinet, Vienne, 1904.  
 L. PIERCE CLARK and THOMAS P. PROUT. Status Epilepticus: A Clinical and Pathological Study in Epilepsy. *Am. Journ. Insan.*, July 1904, p. 81.  
 WALTER H. BUEHLIG. A Case of Astasia-Abasia associated with Epilepsy. *Am. Journ. Insan.*, July 1904, p. 71.  
 ALT. Die diätetische Behandlung der Epileptiker in Vergangenheit und Gegenwart. *Allg. Ztschr. f. Psychiat.*, Bd. lxi., H. 5, 1904, S. 740.

HEINRICH GERHARTZ, Jr. Zur Bluteserumbehandlung der Epilepsie. *Neurol. Centralbl.*, Sept. 16, 1904, S. 835.

**Neurasthenia.**—CAMPBELL MEYERS. Neurasthenia in some of its Relations to Insanity. *Canadian Journ. of Med. and Sur.*, Aug. 1904.  
KRAEMER. Nervenschwäche und Hysterie. Deutscher Verlag, Berlin, 1904, M. 1.

**Hysteria.**—WEILL. Le Développement de l'hystérie dans l'enfance. Steinheil, Paris, 1904.

W. L. ASCHERSON. On Some Distinctions between Hysteria and Neurasthenia, and on their Associations with other Diseases. *Practitioner*, Sept. 1904, p. 353.

RAIMANN. Die hysterischen Geistesstörungen. F. Deuticke, Wien, 1904, M. 9.

RAECKE. Hysterisches Irresein. *Allg. Ztschr. f. Psychiat.*, Bd. lxi., H. 5, 1904, S. 752.

SEIFFER. Hysterische Skolices bei Unfallkranken. A. Hirschwald, Berlin, 1904, M.—50.

BRANDWEINER. Multiple neurotische Hautgangrän. *Monatshefte f. prakt. Dermatol.*, Bd. xxxvii., No. 5, 1904, S. 241.

D. LATTE. Über multiple neurotische Hautgangrän. *Monatshefte f. prakt. Dermatol.*, Bd. xxxvii., No. 4, 1904, S. 159.

W. A. MEARNS. Case of Spontaneous Gangrene of the Skin in a Hysterical Female. *Lancet*, Sept. 17, 1904, p. 824.

SEELIG. Vorstellung eines Kranken im hysterischen Dämmerzustande. *Allg. Ztschr. f. Psychiat.*, Bd. lxi., H. 5, 1904, S. 771.

KLIPSTEIN. Vorstellung eines Kranken mit hysterischen Dämmerzuständen. *Allg. Ztschr. f. Psychiat.*, Bd. lxi., H. 5, 1904, S. 769.

HENRY MEÏGE. La procession dansante d'Echternach. *Nouv. Icon. de la Salpêtrière*, T. xvii., No. 4, 1904, p. 322.

A. L. HUSSEY. On Night-Terrors and Sleep-Walking. *Brit. Journ. Children's Dis.*, Sept. 1904, p. 396.

**Tetany.**—CURSCHMANN. Ueber Pseudotetanie und Uebergangsformen zwischen genuiner und hysterischer Tetanie. *Berl. klin. Wchnschr.*, Sept. 19, 1904, S. 997.

**Tetanus.**—LABADIE-LAGRAVE et LAUBRY. Considérations pathogéniques et thérapeutiques sur un cas de tétanos chronique. Maurin, Paris, 1904.

DEMONTEROT. De la forme paraplégique dans le tétanos chronique. *Thèse*. Baillière et fils, Paris, 1904.

**Myasthenia Gravis.**—STERLING. Kasuistische Beiträge zum Kapitel der asthenischen Paralyse. *Monatschr. f. Psychiat. u. Neurol.*, Bd. xvi., Ergänzungsheft, Aug. 1904, S. 165.

P. K. PEL. Myasthenia pseudo-paralytica (Erb'sche Krankheit) mit Zungenatrophie nach Ueberanstrengung. *Berl. klin. Wchnschr.*, Aug. 29, 1904, S. 917.

J. DE LÉON. Contribution à l'étude de la paralysie myasthénique. *Nouv. Icon. de la Salpêtrière*, T. xvii., No. 4, 1904, p. 269.

**Writer's Cramp.**—ZABLUDOWSKI. Zur Prophylaxe und Therapie des Schreib- und Musikkrampfes. A. Hirschwald, Berlin, 1904, M.—60.

**Tic Convulsif.**—VACLAV PLAVEC. Tic Convulsif. *Wien. med. Presse*, No. 34, 1904, S. 1622.

**Reflex Neuroses.**—G. HUDSON MAKUEN. Reflex Neuroses of Nasal Origin. *Laryngoscope*, St Louis, July 1904.

A. L. BENEDICT. Aesophageal Neuroses. *Am. Journ. Med. Sc.*, Aug. 1904.

EMIL MAYER. Neuroses of the Larynx. *Laryngoscope*, St Louis, July 1904.

F. E. HOPKINS. Neuroses of the Pharynx. *Laryngoscope*, St Louis, July 1904.

**Exophthalmic Goitre.**—M. A. BLISS. Exophthalmic Goitre. Report of a Case. *Med. Fortnightly*, 24, 1904, p. 688.

GOEBEL. Die Basedow'sche Krankheit (Glotsaugenkrankheit) und ihre Behandlung. Verlag der ärztl. Rundschau, München, 1904, M. 1.

GEORGE R. MURRAY. Note on the Serum Treatment of Exophthalmic Goitre. *Lancet*, Aug. 27, 1904, p. 583.  
M. FIGUEIRA. Excision of the Thyroid in Graves' Disease. *Brooklyn Med. Journ.*, Aug. 1904.

**Cretinism.**—BAYON. Ueber die Aetiologie des Schilddrüsenschwunds bei Cretinismus und Myxödem. *Neurol. Centralbl.*, Sept. 1, 1904, S. 792.  
F. J. KIEFFER. Cretinism. Synonyms: Sporadic Cretinism, Cretinoid Idiocy, Myxoedematous Idiocy, with Pachydermatous Cachexia. *Amer. Practitioner and News*, July 15, 1904.  
WEYGANDT. Weitere Beiträge zur Lehre vom Cretinismus. A. Stuber, Würzburg, 1904, M. 3.

**Adiposis Dolorosa.**—J. N. HALL and C. E. Walbrach. Adiposis Dolorosa. With Report of Three Cases. *Am. Journ. Med. Sc.*, Aug. 1904.  
DELUCQ et ALAUX. Adipose douloureuse (mal de Deroum). *Presse med.*, sept. 17, 1904, p. 593.

#### ALCOHOLISM, POISONS, ETC.—

ALZHEIMER. Ueber das Delirium alcoholicum febrile Magnana. *Monatschr. f. Psychiat. u. Neurol.*, Bd. xvi., H. 2, 1904, S. 285.  
E. KNECHT. Zur Kenntnis der Erkrankungen des Nervensystems nach Kohlenoxydvergiftung. *Deutsche med. Wchnschr.*, Aug. 18, 1904, S. 1242.  
CHARLES J. DOUGLASS. Morphinism: its Symptomatology. *New York Med. Journ.*, Aug. 13, 1904.

#### SPECIAL SENSES AND CRANIAL NERVES—

GALEZOWSKI. Le Fond de l'œil dans les affections du système nerveux. *Thèse. F. Alcan, Paris*, 1904, 5 fr.  
HERBST. Papillitis und Amenorrhoe. *Wien. klin. Wchnschr.*, Sept. 15, 1904, S. 990.  
ABRAHAM. Vorstellung eines Kranken mit Hemianopsie und Rotgrünblindheit im erhaltenen Gesichtsfeld. *Centralbl. f. Nervenheilk. u. Psychiat.*, Sept. 15, 1904, S. 578.  
JULIUS DONATH. Pupillenprüfung und Pupillenreaktion. *Monatschr. f. Psychiat. u. Neurol.*, Bd. xvi., Ergänzungsheft, Aug. 1904, S. 191.  
J. PILTZ. Ein neuer Apparat zum Photographieren der Pupillenbewegungen. *Neurol. Centralbl.*, Sept. 1, 1904, S. 801.  
VOLHARD. Ueber Augensymptome bei Armlähmungen. *Deutsche med. Wchnschr.*, Sept. 8, 1904, S. 1339.  
A. MARINA. Ueber die bei den coordinirten Seitenbewegungen des Augen an dem sich medialwärts drehenden Auge eintretende Pupillenverengung. *Neurol. Centralbl.*, Sept. 1, 1904, S. 797.  
ROCHE. Contribution à l'étude des paralysies oculaires traumatiques d'origine orbitaire. *Thèse. Roussel, Paris*, 1904.  
HARVEY CUSHING. The Sensory Distribution of the Fifth Cranial Nerve. *Bull. Johns Hopkins Hospital*, July-Aug. 1904, p. 213.  
H. de L. SPENCER. Report of Two Cases of Hemifacial Atrophy, with presentation of Patients. *Cleveland Med. Journ.*, July 1904.  
STEGMANN. Ein Fall von Atrophia faciei und seine kosmetische Behandlung. *Wien. klin. Wchnschr.*, Sept. 1, 1904, S. 944.  
LAPERSONNE. Le Syndrome oculaire neuro-paralytique. Germain et Grassin, Angers, 1904.  
LEOPOLD. Kasuistischer Beitrag zur Kenntnis des Herpes zoster ophthalmicus. F. Pletscher, Tübingen, 1904, M.—70.  
C. ZIMMERMANN. Affections of the Facial Nerve in Diseases of the Ear. *Arch. of Otol.*, Vol. xxxiii., No. 4, 1904, p. 290.  
SCHEIBER. Beitrag zur Lehre von der Thränensekretion im Anschlusse an drei Fälle von Facialislähmung mit Thränenmangel, nebst Bemerkungen über den Geschmackssinn und über Sensibilitätsstörungen bei Facialislähmungen. *Deutsche Ztschr. f. Nervenheilk.*, Bd. xxvii., H. 1 u. 2, 1904, S. 45.  
MAX MANN. Über Gaumenlähmung. *Ztschr. f. Ohrenheilk.*, Bd. xlvii., H. 4, 1904, S. 1.  
M. ROSE. Paralyse des nerfs crâniens d'un côté et déformations osseuses multiples d'origine probablement héredo-syphilitique tardive. *Nouv. Icon. de la Salpêtrière*, T. xvii., No. 4, 1904, p. 277.

## MISCELLANEOUS SYMPTOMS—

- Z. BYCHOWSKI. Ueber Hypotonie und Hypertonie bei einer und derselben Kranken. *Neurol. Centralbl.*, Sept. 1, 1904, S. 786.
- ST CLAIR B. SHADWELL. Tender Spots on the Spine in relation to Pain in Various Parts of the Body. *Lancet*, Sept. 10, 1904, p. 754.
- SEIDELMANN. Ein Fall von symmetrischer Gangrän der Extremitäten nach Pneumonie. *Deutsche Ztschr. f. Nervenheilk.*, Bd. xxvii, H. 1 u. 2, 1904, S. 114.
- SOUKHANOFF. Sur le syndrome de Ganser ou le symptomo-complexus des réponses absurdes. *Rev. Neurol.*, sept. 15, 1904, p. 933.
- HENNEBERG. Das Gansersche Symptom. *Allg. Ztschr. f. Psychiat.*, Bd. lxi, H. 5, 1904, S. 735.
- SAINTON et VOISIN. Le signe de Kernig. *Gaz. d. hôp.*, août 27, 1904, p. 947.
- F. W. MOTT. Tremors. *Practitioner*, Sept. 1904, p. 293.
- BOUCHAUD. Un cas d'anesthésie avec troubles de la sensibilité objective à topographie segmentaire. *Rev. Neurol.*, août 15, 1904, p. 826.
- BUNHOEFFER. Der Korsakowsche Symptomenkomplex in seinen Beziehungen zu den verschiedenen Krankheitsformen. *Allg. Ztschr. f. Psychiat.*, Bd. lxi, H. 5, 1904, S. 744.
- JOHN LINDSAY STEVEN. A Case of General Amnesia? *Glasgow Med. Journ.*, Sept. 1904, p. 161.
- EMMA W. MOORS. A Case of Syphilis of the Nervous System presenting Clinically an Amnesic Symptom Complex, with Autopsy. *Am. Journ. Insan.*, July 1904, p. 11.

**Aphasia.**—DEJERINE et THOMAS. Contribution à l'étude de l'aphasie sensorielle. *Rev. Neurol.*, août 15, 1904, p. 805.

GUERIDO. L'Aphasie amnésique. *Thèse*. Firmin, Montane et Sicardi, Montpellier, 1904.

SYDNEY STEPHENSON. Congenital Word-Blindness. *Lancet*, Sept. 17, 1904, p. 827.

**Reflexes.**—MARIO BERTOLOTTI. Le condizioni della presenza del segno di Babinski nei casi di lesione extrapiramidale. *Riv. di Patol. nerv. e ment.*, Vol. ix, f. 9, 1904, p. 430.

F. JAMIN. Ueber das Verhalten der Bauchdeckenreflexe bei Erkrankungen der Abdominalorgane. *Monatschr. f. Psychiat. u. Neurol.*, Bd. xvi, H. 2, 1904, S. 289.

W. V. BECHTEREW. Ueber den Glutäalreflex. *Neurol. Centralbl.*, Sept. 16, 1904, S. 833.

ALEXANDER WILSON. The Respiratory and Cardiac Reflex induced by Peripheral Impressions on the Pudic Nerve during Anesthesia. *Lancet*, Aug. 20, 1904, p. 521.

## MENTAL DISEASES—

ANGLADE et JACQUIN. Rapport médical de l'asile public d'aliénés de Bordeaux pour l'année 1903. Imp. Gounouilhoul, Bordeaux, 1904.

MAX HACKL. Das Anwachsen der Geisteskranken in Deutschland. Seitz & Schauer, Münschen, 1904, M. 3.

J. B. LOUGHARY. Increase of Insanity and its Remedy. *North-West Med.*, Seattle, July, 1904.

HOCHE. Einteilung und Benennung der Psychosen mit Rücksicht auf die Anforderungen der ärztlichen Prüfung. *Allg. Ztschr. f. Psychiat.*, Bd. lxi, H. 5, 1904, S. 787.

MITTENZWEIG. Hirngewicht und Geisteskrankheit. *Allg. Ztschr. f. Psychiat.*, Bd. lxi, H. 5, 1904, S. 773.

SOMMER. Experimentelle Psychopathologie. *Allg. Ztschr. f. Psychiat.*, Bd. lxi, H. 5, 1904, S. 785.

HANS BERGER. Experimentelle Studien zur Pathogenese der Geisteskrankheiten. *Monatschr. f. Psychiat. u. Neurol.*, Bd. xvi, H. 2, 1904, S. 213.

WILLIAM RUSH DUNTON, Jr. Some Observations on Blood Pressure in the Insane. *Am. Journ. Insan.*, July 1904, p. 41.

SCHULTZE. Beziehungen zwischen chemischer Konstitution und hypnotischer Wirkung. *Allg. Ztschr. f. Psychiat.*, Bd. lxi, H. 5, 1904, S. 743.

MASSELON. Un cas de forme fruste de démence précoce. *Hérissey, Evreux*, 1904.



- MEEUS. Considérations générales sur la signification clinique de la démence précoce. *Ann. méd.-psychol.*, Sept.-Oct. 1904, p. 207.
- DENY. Psychiatrie. Des démences vésaniques. Empéreaux, Pau, 1904.
- GINO MONZARDO. Contributo allo studio delle inversioni sessuali. *Riv. mens. di psichiat. forense*, Anno vii., Num. 7-8, 1904, p. 286.
- A. LA CARA. Su di un caso di libidine sanguinaria. *Riv. mens. psichiat. forense*, Anno vii., Num. 7-8, 1904, p. 280.
- SKLAREK und van VLEUTEN. Gleichzeitig bei drei Geschwistern aufgetretene geistige Erkrankung. *Allg. Ztschr. f. Psychiat.*, Bd. lxi., H. 5, 1904, S. 775.
- GIERLICH. Ueber periodische Paranoia. *Monatschr. f. Psychiat. u. Neurol.*, Bd. xvi., H. 2, 1904, S. 286.
- WEBER. Ueber akute tödlich verlaufende Psychosen. *Monatschr. f. Psychiat. u. Neurol.*, Bd. xvi., Ergänzungsheft, Aug. 1904, S. 81.
- JOHN W. STEVENS. Manic-Depressive Insanity, with the Report of a Typical Case. *Journ. Nerv. and Ment. Dis.*, Aug. 1904, p. 513.
- F. QUENSEL. Neue Erfahrungen über Geistesstörungen nach Schwefelkohlenstoffvergiftung. *Monatschr. f. Psychiat. u. Neurol.*, Bd. xvi., H. 2, 1904, S. 246.
- SCHÜLE. Nochmals das Heiraten von früher Geisteskranken. *Allg. Ztschr. f. Psychiat.*, Bd. lxi., H. 5, 1904, S. 761.
- B. FRANK TURNER. The Diagnosis of Insanity. *Memphis Med. Monthly*, July 1904.
- HOPPE. Simulation und Geistesstörung (Schluss). *Vierteljahrsschr. f. gericht. Med.*, Juli 1904, S. 91.
- L. THIVET. Simulation et dissimulation de la folie. *Ann. méd.-psychol.*, Sept.-Oct. 1904, p. 236.
- N. TOPORKOFF. Une tentative d'analyse psychologique des travaux manuels des aliénés. *Nouv. Icon. de la Salpêtrière*, T. xvii., No. 4, 1904, p. 311.
- CAREY B. GAMBLE. Mental Phenomena and Visceral Disease. *Bull. Johns Hopkins Hospital*, July-Aug. 1904, p. 247.
- VURPAS. L'état moteur des aliénés. *Rev. de Psychiat.*, août 1904, p. 309.
- W. G. MELVIN. On the Influence of Mode of Life upon the Blood of Inmates of a Hospital for the Insane. *Am. Journ. Insan.*, July 1904, p. 109.
- CH. FÈRE et MLLÉ. A. MOUROUX. Note sur la fréquence et sur la distribution des nevri chez les aliénés. *Arch. de Neurol.*, sept. 1904, p. 193.
- DEXLER. Ueber die psychotischen Erkrankungen der Tiere. *Monatschr. f. Psychiat. u. Neurol.*, Bd. xvi., Ergänzungsheft, Aug. 1904, S. 99.
- SIEGFRIED TÜRKEL. Die kriminellen Geisteskranken. *Wien. med. Presse*, No. 38, 1904, S. 1766.
- LOUISE G. ROBINOVITCH. Suicidal and Homicidal Acts: their Clinical Aspects and Medico-Legal Significance. *Journ. Ment. Pathol.*, Vol. v., Nos. 4-5, 1904, p. 187.
- VENTRA e ANGIOLELLA. Un caso di frenosi sensoria in rapporto ad imputazione di falsi e peculato. *Manicomio*, Anno xx., No. 2, 1904, p. 238.
- P. PENTA. Simulazione di pazzia e reali disturbi o difetti psichici nei criminali. *Riv. mens. di psichiat. forense*, Guigno, 1904, p. 217.

## TREATMENT—

- J. J. PUTNAM. A Consideration of Mental Therapeutics as employed by Special Students of the Subject. *Boston Med. and Surg. Journ.*, Aug. 18, 1904, p. 179.
- D. M. HALL. Faith and Suggestion in the Practice of Medicine. *Memphis Med. Monthly*, July 1904.
- JAMES SHERRIN. A Lecture on the Treatment of Common Nerve Injuries. *Clin. Journ.*, Aug. 31, 1904, p. 311.
- GARBINI e VITALI. Sull' azione ipnotica e sedativa del Veronale nei malati di mente. *Manicomio*, Anno xx., No. 2, 1904, p. 153.
- QUENÉ. Traitement de l'urémie nerveuse par la ponction lombaire (thèse). Morel, Lille, 1904.
- L. PIERCE CLARK and H. P. ALAN MONTGOMERY. Suggestions and Plans for Psychopathic Wards, Pavilions, and Hospitals for American Cities. *Am. Journ. Insan.*, July 1904, p. 1.
- WILLIAM P. SPRATLING. The Principles of Colony Building for the Defective Classes. *Am. Journ. Insan.*, July 1904, p. 77.
- OWEN COPP. Characteristics of the Scotch Lunacy System. *Am. Journ. Insan.*, July 1904, p. 55.

**KÉRAVAL.** *Assistance.* Des mesures à prendre à l'égard des aliénés criminels. Empérauger, Pau, 1904.

**C. WICKEL.** Family Care of the Insane. *Med. Press*, Sept. 21, 1904, p. 304.

**Surgical.**—**ROQUEPLO.** De la craniectomie au moyen de la scie de Gigli. Firmin, Montane et Sicardi, Montpellier, 1904.

**G. C. MACDONALD.** Surgical Aspect of a Case of Jacksonian Epilepsy. *Am. Med.*, Aug. 6, 1904.

**ROYAL WHITMAN.** Remarks on the Surgical Treatment of Obstetrical Paralysis. *Journ. Nerv. and Ment. Dis.*, Aug. 1904, p. 487.

**LAFOY.** Contribution à l'étude de l'extraction des balles intracrâniennes. Thèse, Baillière et fils, Paris, 1904.

**J. JACKSON CLARKE.** Paralytic Deformities and their Modern Treatment. *Practitioner*, Sept. 1904, p. 393.

**WILLIAM SHEEN and WILLIAM MARTIN.** A Case of Bullet Wound of the Brain, in which the Bullet Lodged about 12 centimetres from the Point of Entrance, and was Extracted from a Depth of 6·5 centimetres from the Surface of the Head. *Lancet*, Sept. 17, 1904, p. 825.

**A. CANGÉ.** Déformation singulière et symétrique des avant-bras et des mains ; résection orthopédique ; guérison. *Nouv. Icon. de la Salpêtrière*, T. xvii., No. 4, 1904, p. 283.

# Review of Neurology and Psychiatry

---

## Original Articles

### **A CASE OF INTRA-CRANIAL ANEURISM IN A YOUNG SUBJECT.**

By ALEXANDER BRUCE, M.D., F.R.C.P.Ed.,

and

W. B. DRUMMOND, M.B., C.M., F.R.C.P.Ed.

ROBERT BOSWELL, a miner, aged 27 years, was admitted to Ward VI. of the Royal Infirmary on the afternoon of January 22nd, 1898. He was brought to the Infirmary by the police, having taken a fit in the street. When admitted he was just recovering consciousness, and it was noted that he had been vomiting. Very soon after admission he took another fit of so severe a character that the nurse who attended him thought he was going to die. After the fit the patient became very violent. At 10 P.M. he had 60 grs. of bromide of potassium, and slept for two hours, after which he again became extremely restless, and threw himself about in bed so violently that mechanical restraint had to be resorted to in order to prevent him from injuring himself.

Before describing further the condition of the patient on admission, such facts as were afterwards ascertained concerning his previous history may be narrated. His father and mother both died when he was about three years of age; his father of consumption, his mother of ulceration of the stomach—suddenly. He had no sisters; his only brother died at the age of eighteen months. Patient was brought up by an uncle, who describes him

as having always been healthy and of very temperate habits. About a year before admission his uncle thought he was not looking well, and advised him to stop work for a little. This he did, and apparently with some benefit to his bodily health; but he became dull and irritable and disinclined for his work, to which he never returned. He does not seem to have complained much of any special symptoms, but during the month before admission he did complain occasionally of vomiting after food, and on one or two occasions he also suffered from headache, to which he had not previously been subject.

As to the condition of the patient on admission, it was noted that he was a well-developed and fairly well-built dark-complexioned man. His general nutrition was good. The backs of his hands were "tattooed" with specks of coal dust, but his hands were soft, as if he had not been at work for some time.

After the excitement from which he was suffering had subsided, he complained greatly of intense headache, "as if his head would burst," and often assumed the knee-elbow position, boring his head into the pillow. He answered questions rationally, but his memory was considerably impaired, especially for recent events. He remembered coming into town on Saturday (the day of admission), but maintained that he had been at work till quite lately—sometimes saying that he had not been off work at all, at others that he had been off for a fortnight only. His sensibility to touch, muscular sense, etc., were normal. The knee-jerks and cutaneous reflexes were normal. There was no vomiting, but the ward attendant stated that there was sometimes after a meal some regurgitation of food into the mouth, which was again swallowed. Urine and feces were passed in bed, the patient stating that he was unconscious of their passage.

There was no paralysis anywhere.

The pupils were equal, and reacted to light and accommodation. There was no nystagmus. The optic discs, examined a day or two after admission, were found to be somewhat reddened, with diminution of the physiological cup, but the vessels could be clearly traced into the disc. Dr Sym examined the discs on the 29th, and reported that there was considerable venous engorgement of both discs, especially of the left, but that the changes did not amount to optic neuritis.

With regard to the other symptoms, it need only be noted that the lungs were healthy; the vascular system appeared to be normal, especially there was no evidence of endocarditis; the urine contained a trace of albumen at the time of admission. This had disappeared the next day, and thereafter the urine continued normal.

On the day after admission, then, and for fully a week thereafter, the most prominent feature in the case was the intense headache from which the patient suffered, headache so intense that he was often found in the knee-elbow position, pressing his forehead into the pillows; so intense that every now and again he was seized by attacks of momentary frenzy, during which he would plunge violently about in the bed, or seize any small object near him, such as the ice-bag which had been placed on his head, and throw it across the ward. The pain was of a "bursting" character, not localised to any particular site, but said to be worst over the vertex and at the back of the head. There was some diffused tenderness over the scalp from the vertex to the external occipital protuberance. There was also slight tenderness, ill-defined, at the back of the neck.

The only other features of any importance during this time were occasional slight regurgitation of food into the mouth, slight congestion of the optic discs, and involuntary passage of urine and faeces.

Many things were tried for the relief of the headache. Morphia alone had any marked effect.

On the 29th of January patient complained of occasionally seeing double when he first looked at an object, but this passed off quickly when he continued to look at it. No strabismus was visible.

On the 30th there was evident weakness of the internal and to a slighter extent of the external rectus of the right eye. The patient complained that moving his eye caused great pain. Movement of the head caused great pain in the back of the neck, and there was considerable tenderness on pressure over the upper part of the posterior triangles. There was no change in the optic discs.

On the following day the paresis of the right internal and external recti was well marked, there was slight impairment of the upward and downward movements of the eye, and also slight ptosis.

During the next few days the patient seemed to improve somewhat. The headache became less severe, the incontinence passed off, and the paresis of the right III. and VI. nerves became less marked.

On the 6th, Dr Sym again examined the optic discs and reported that the congestion was subsiding.

The temperature since admission had been slightly febrile, varying between  $99^{\circ}$  and  $100.5^{\circ}$ ; since February 1st the temperature has been normal.

These notes may terminate the record of the first period of the patient's illness subsequent to admission. The symptoms narrated seemed to indicate the presence of some gross lesion, probably a tumour, associated possibly with some basal meningitis. As to the possible nature of the tumour, if such existed, there was nothing to guide us. No history of syphilis had been obtained, there was no evidence of vascular degeneration (the patient was only twenty-seven years of age) nor of valvular disease of the heart, whose frequent association with cerebral aneurism has been noted by Gowers. On the 7th of February, then, the patient appeared to be improving decidedly, but that evening the severe headache (for some days in abeyance) returned suddenly in an aggravated form—the patient exclaimed "I never felt pain till now"—and about twenty minutes later (8.20 P.M.) he took an epileptiform convulsion. After this he never recovered complete consciousness. A second convulsion occurred at 9.15, and a third at 9.50. Between the fits the head and eyes were turned strongly to the left. During the night numerous attacks of spasm occurred. For some hours these occurred about every five minutes, each period of spasm lasting about forty or fifty seconds. The spasm began with a gurgling sound in the throat; the breathing became accelerated (to 52 per minute) and noisy; and the pulse to 120 or 130. The temperature rose (at 12.30) to  $103^{\circ}$ . The limbs became rigid; the arms were extended in front of the body and rotated so that the hands were placed back to back. The fingers were closed. The intercostal muscles and diaphragm acted violently, and it was noted that the left cheek was puffed out. In the intervals between the spasms the breathing was comparatively quiet and tranquil. All the limbs were quite placid. The left knee-jerk was slightly exaggerated, the right was

practically absent. The pupils varied in size and reacted sluggishly to light.

The patient died at 7 A.M.

NOTES ON POST-MORTEM EXAMINATION, FEB. 8, 1898.

Body slightly emaciated but well developed and muscular. Rigidity well marked. No morbid appearances.

*Head.*—Dura mater tense. Superficial veins congested. Superior longitudinal sinus contains ordinary clot.

Convolutions somewhat flattened on both sides; a small quantity of recent clot lying free over both frontal lobes.

At the base of the brain there was extensive diffuse sub-arachnoid hæmorrhage, extending from the frontal region, covering the optic chiasma and the whole of the anterior surface of the pons and medulla, also passing round to the cerebellum, as well as at the beginning of the Sylvian fissures. On opening the brain there was found a considerable quantity of recent coagulum in both lateral ventricles, but more abundant in the left. The blood had not passed down into the third ventricle. On separating the frontal lobes there was found a spherical aneurism about the size of a small hazel-nut. This was situated immediately in front of the anterior communicating artery, and its interior was connected with that of the latter by a small opening. The anterior cerebral arteries ran over the surface of the sac, one on each side, and were closely applied to it.

The aneurism was in great part filled with fine red laminated clot. It had burst on the left side, and there was considerable softening and infiltration of the tissues of the left frontal lobe extending forward nearly to its anterior end. The softened brain substance had a dark brownish to yellow colour. There had accordingly been for some time a slow oozing of blood from the sac, and more recently a larger rupture had occurred, the blood passing both upwards into the left lateral ventricle, and also into the subarachnoid space, where it had become widely diffused. The hæmorrhage could be traced directly upwards from the aneurism to the ventricle.

There was considerable matting of the membranes at the base, both between the frontal lobes and also at the commencement of the Sylvian fissures, especially on the left side—a condition of chronic meningitis.

The walls of the arteries at the base elsewhere appeared quite healthy.

Only the head was examined.

---

## THE MODE OF TURNING, IN WALKING, OF ORGANIC HEMIPLEGICS AND SPASTIC PARAPLEGICS.

By LEONARD J. KIDD, M.D.

THERE is a reference in the *Journal of Nervous and Mental Disease* for July 1903, under the heading of "Flank Gait," to a paper by von Schüller, in the *Neurol. Centralblatt*, 1903, vol. xxii., No. 2, January 16th, in which he says that "an organic hemiplegic patient can walk sideways towards his paralysed side with little or no difficulty, but on account of the spasticity and lengthening of the palsied leg he has much difficulty in walking sidewise in the opposite direction, and must drag the paralysed leg."<sup>1</sup>

It is also pointed out that the functional hemiplegic cannot walk towards the palsied side, but only to the sound side.

As this test takes time, and rather disturbs the patient's mind, and adds to the already long list of those needed in clinical neurology, I determined I would test the point more easily and quite as effectually by watching to which side the patient turned round in walking.

I have now watched some hundreds of cases of organic hemiplegia and spastic paraplegia with this object in view, and in fully 99 per cent. the patient has spontaneously turned to the spastic or more spastic side when told to walk to the end of the room, turn round, and come back.

In many of the cases the gait was tested thus two or more times; in no case did the patient turn to the opposite side to that chosen at his first trial.

In the very small minority (less than 1 per cent. probably) there was not in a single case the slightest doubt, from a consideration of other physical signs, that the condition was organic.

<sup>1</sup> Quotation here is from *Journ. Nerv. and Ment. Dis.*



I have never seen a functional hemiplegic turn round to the affected side.

So that, as far as my one year's experience on this point goes, I look upon von Schüller's phenomenon as a diagnostic point of the highest importance.

I think I shall not be going in excess of the facts if I enunciate the following propositions:—

(1) If a spastic hemiplegic or a spastic paraplegic turns round in walking to the affected or the more affected side, an organic lesion is present.

(2) If he turns to the sound or less affected side, the case is either one of functional or of organic affection. If the latter, there will be no difficulty in deciding in its favour by a careful physical examination.

A point of much interest and of possible importance arises out of my observations.

In the few cases (five or six) I have seen during the last twelve months of what is often called "Hemichorea," the patient in every instance turned to the affected side.

I think that von Schüller's phenomenon may have a much wider range than that of hemiplegia, and that a wider and longer experience will very likely show that in cases of unilateral weakness of organic origin the patient usually turns to the weaker side.

I cannot too strongly urge clinicians to watch, in testing the gait of all nervous cases, to which side the patient turns, especially in those that show unilateral weakness, or spasm, or stiffness.

Observations are needed also to decide whether flaccid organic hemiplegias follow the rule of the spastic cases.

I think it is better to defer any attempt to explain the physiological mechanism of v. Schüller's phenomenon and its few exceptions to a future paper, as the matter is somewhat theoretical and may lead to controversy; the important thing now is that clinicians should test the practical point, and learn to value its enormous diagnostic value.

In conclusion, I most cordially return my thanks to the physicians to out-patients, and to the assistant physicians at the National Hospital, Bloomsbury, for the privilege of seeing their cases, which have been the main source of my observations.

# Abstracts

## ANATOMY.

### THE MAMMALIAN CEREBELLUM: ITS LOBES AND FISSURES.

(364) O. CHARNOCK BRADLEY, *Journ. Anat. and Phys.*, Vol. xxxviii. p. 448, and Vol. xxxix. p. 99.

THE present paper forms a continuation of a previous communication to the same journal, an abstract of which appeared in this Review (Vol. i. p. 482). Sheep, calf, horse and human embryos, and a number of adult cerebella, mainly of Primates, have been examined.

The author is of opinion that the cerebellum of the Anthro-  
poidea in many respects stands apart from that organ of the rest  
of the mammalia. There is a graded series of forms commencing  
in the Hapalidæ and culminating in man. Fissure I. is deep in all  
monkeys, and in most monkeys lobule A<sub>1</sub> is, relatively speaking,  
small, whereas lobe B is well developed. In lobe C there is a  
gradual increase in size from the Hapalidæ upwards. Lobule D<sub>1</sub>  
is characterised by a remarkable constancy in size of those parts  
of it which are included in the hemisphere. The fairly uniform  
size of the flocculus and of lobe E in general is noteworthy. In  
monkeys, too, the paraflocculus is fairly uniform, and is peculiar  
in that it consists of only one well-developed row of folia.

The arbor vitæ cerebelli consists of a central white mass, more  
or less divided into two parts, from which two strong main  
branches spring (the *truncus verticalis* and *truncus horizontalis*  
of Ziehen). In all but the simplest forms of cerebella there are  
five prominent branches in addition to the two main trunks. The  
seven branches so produced are distributed to the following lobes  
and lobules:—(1) to lobule A<sub>1</sub>, (2) to lobule A<sub>2</sub>, (3) to lobe B, (4)  
to lobe C, (5) to lobule D<sub>1</sub>, (6) to lobule D<sub>2</sub>, and (7) to lobe E.

Of the five lobes—A, B, C, D and E—into which the cere-  
bellum can be divided, A and B (the most anterior) differ from the  
rest in possessing shallow paramedian sulci. In the earlier stages  
of development, also, they lag behind the others. Later, however,  
they out-distance the posterior lobes in development, and so come  
to resemble the adult lobes, while C, D and E are still compara-  
tively rudimentary.

Lobe C occupies a developmental and anatomical position  
peculiarly its own. Upon the marked capacity of its two posterior  
lobules to grow in a sagittal direction depends the most striking  
differences to be observed in a series of mammalian cerebella.

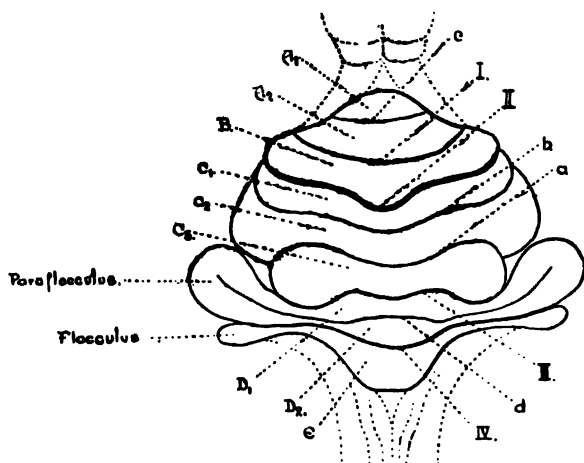


FIG. 1.—Scheme of the Lobes and Lobules of the Mammalian Cerebellum arranged in one plane.

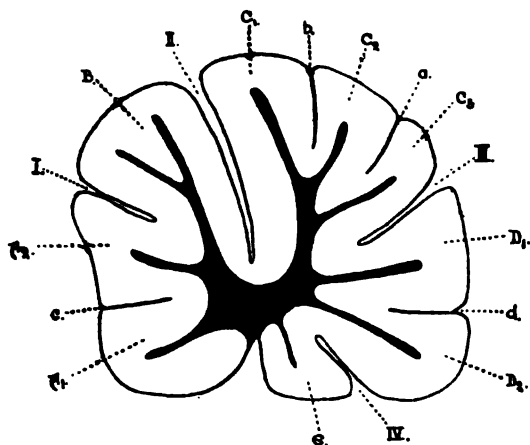


FIG. 2.—Scheme of the Mesial Sagittal Section of the Average Mammalian Cerebellum.

This sagittal expansion is most marked in the hemispheres, but is possible, generally in a lesser degree, in the vermis as well.

Lobule  $D_1$  has an outlying territory in the dorsal parafocculus, with which the vermis may or may not be directly connected. Lobule  $D_2$  is simpler than  $D_1$ ; the ventral parafocculus, embryologically connected with the vermis, is generally isolated in the adult.

Lobe E is notably constant in size. It consists of the nodulus and flocculus, the connection between which is frequently lost.

The difference in the size of the parafocculus in different mammals is very striking. In its most highly developed condition it is composed of two rows of folia (*dorsal* and *ventral parafocculus* of Elliot Smith), to which may or may not be added a *lobulus petrosus*. In the apes the parafocculus shows various grades of diminution; and in man it has entirely disappeared in many, if not most, cases.

AUTHOR'S ABSTRACT.

**THE COLLATERAL SULCUS.** E. ZUCKERKANDL, *Arch. a. d.* (365) *Neurol. Instit. Wien*, Bd. xi, 1904, p. 407.

THIS is an account of the comparative anatomy of the collateral and occipito-temporal sulci in the apes and man.

In the lowlier apes a furrow makes its appearance on the mesial surface of the hemisphere below the caudal part of the calcarine [retrocalcarine] sulcus: it gradually extends *forward* toward the rhinal fissure. This is the collateral sulcus.

A second furrow makes its first appearance immediately to the outer side of the rhinal fissure and grows *backward* on to the tentorial, or in some cases the lateral aspect of the hemisphere, i.e. external to the collateral sulcus. It is the occipito-temporal. Several writers, Ziehen and Flatau and Jacobsohn among the number, compared these two sulci, and called the *lateral* one in the lowlier apes by the name collateral.

The memoir gives a detailed account of the variations which these two sulci present in a large series of apes. The author does not seem to be aware of the fact that I covered most of the ground traversed in his elaborate memoir in the notes published two years ago in the Descriptive Catalogue of the Royal College of Surgeons of England. G. ELLIOT SMITH.

**AN APPARATUS FOR ESTIMATING THE VOLUME OF THE** (366) **BRAIN—CEREBROVOLUMINIMETER.** F. REICH, *Neurolog. Centralbl.*, Nr. 18, 1904, p. 839.

THE determination of the size of the brain by weighing, the method chiefly in use hitherto, is unreliable, because brains of the same

volume may have dissimilar weights, and therefore comparison of the relative sizes of brain and cranial cavity, the only mode of ascertaining whether cerebral atrophy has or has not taken place, is impossible. Richter, however, carried out a number of careful determinations of volume by placing the brain in a graduated glass vessel partly filled with water, and reading off the height of the latter before and after the immersion. Owing to the width of the vessel employed this method is difficult to carry out with accuracy, and therefore the following modification has been adopted and used with success:—The apparatus consists of a cylindrical glass vessel, large enough to hold a brain, and provided at a certain distance from the bottom with an aperture connected with a pipe and tap. The vessel having been filled up to a certain mark with water, salt solution, or Müller's fluid, the tap is opened and the fluid runs away until the level of the aperture is reached. The brain is then immersed, the tap being first closed, and as soon as the fluid is still the tap is again opened, and the fluid this time received into a tall graduated measure of over 2000 c.cm. capacity. The quantity read off as soon as the flow from the large vessel has ceased gives the volume of the brain. The method has been found simple in use and sufficiently accurate, and it does not involve interruption of the autopsy.

W. R. DAWSON.

### PHYSIOLOGY.

**THE PHYSIOLOGICAL AREAS AND CENTRES OF THE CERE-  
(367) BRAL CORTEX OF MAN, WITH NEW DIAGRAMMATIC  
SCHEMES.** C. K. MILLS, *Univ. Penn. Med. Bulletin*, May  
1904.

THE author's chief object is to present his latest views on the entire subject of cerebral localisation in two new diagrams—one of the lateral, and the other of the mesial aspect of the hemi-cerebrum. The positions, both of some of the larger areas and of the sub-areas, have been considerably shifted from positions formerly held. Conformably to the recent researches of Sherrington and Grünbaum, and as a result of personal investigation, the great motor region is now placed entirely in, and in front of the Rolandic fissure, the ascending parietal (post-central) convolution being given over to cutaneous representation. The author warns against referring to a motor zone or area: motor zones or areas is the correct expression of the facts, for each of the special areas of representation of the senses has its corresponding motor area. The motor areas are, therefore, as follows:—

1. The great motor area.
2. The visual motor area.

3. The auditory motor area.
4. The olfactory motor area.
5. The gustatory motor area.

And, if the vestibular nerve have a cortical cerebral representation in an equilibratory centre, we should have

6. An equilibratory motor area.

Remarks follow on each of these motor areas, giving the latest results of experimental and clinico-pathological evidence, though the discussion does not pretend to be by any means exhaustive. The fact that cerebral tumours situated in the occipital and occipito-parietal cortex may reveal visual motor symptoms in the shape of paresis or paralysis of certain eye muscles is emphasised as being of considerable importance from the point of view of localisation. The author further figures definitely a somewhat isolated centre for eye movements anterior to the commonly accepted motor region, in front of and below a uniting centre in the posterior part of the second frontal convolution. S. A. KINNIER WILSON.

**THE OPTIC REFLEX APPARATUS OF VERTEBRATES FOR  
(368) SHORT-CIRCUIT TRANSMISSION OF MOTOR REFLEXES  
THROUGH REISSNER'S FIBRE: ITS MORPHOLOGY,  
ONTOGENY, PHYLOGENY, AND FUNCTION. PART I.  
THE FISH-LIKE VERTEBRATES. PORTER EDWARD  
SARGENT, *Bull. of the Museum of Comp. Zoology at Harvard  
College*, Vol. xlv., No. 3, July 1904, p. 131.**

FORTY-FOUR years ago Reissner described a nerve-fibre lying in the lumen of the central canal of the central nervous system of *Petromyzon*. A long series of other anatomists, both before and after Reissner, have seen this fibre in various vertebrates, but have regarded it as an artefact, either a coagulation mass formed from the cerebro-spinal fluid, or as "thrown-off epithelial cells or blood corpuscles." It remained for Sargent, in a preliminary note published in 1900, to redemonstrate the nervous nature of this structure, and since then his statements have been confirmed by some writers and denied by others.

The present memoir is the first instalment of a great monograph on Reissner's fibre in the vertebrata: it deals with the cyclostomata, selachii, ganoidea, and teleostei, and contains a detailed description of the fibre-tract, its staining reactions, its cell-connections, its developmental history, and its functions in these various fishes.

Immediately behind the posterior commissure there is a group of large cells lying in the mesencephalic roof alongside the mesial

plane—the “*optic reflex cells*”—the axis cylinders of which converge toward the anterior end of the mesocœle or aqueduct of Sylvius, and pass into the ventricular cavity, where they become collected into a compact bundle—Reissner’s fibre. In some forms the fibre also receives some elements from the ganglia habenulæ. Reissner’s fibre passes along the central canal in the caudal direction, and distributes fibres to the posterior two-thirds of the spinal cord.

One set of processes from the “optic reflex-cells” crosses through the dorsal decussation, and in the lateral part of the tectum it breaks up in a superficial position among the terminal processes of the optic nerve-fibres. Another set passes backward in the tectum to end in the cerebellum. “When Reissner’s fibre is severed the power to respond quickly to optical stimuli is lost” (p. 231).

“Reissner’s fibre apparatus may be a great time-saver. It is evident that this short circuit may mean the saving of a considerable fraction of a second. In the struggle for existence an animal may be frequently presented with ocular evidence of danger, the only safety from which lies in flight, dodging, or some other quick reaction from the source of danger. A decrease of a small fraction of a second in the reaction-time may often be a matter of escape or injury, of life or death, and so the determining factor in the survival of the fittest individual or species” (p. 240).

The author promises us an account of this apparatus in the higher vertebrates, and a general discussion of the significance of his results at some time during the next twelve months.

There is an immense amount of information concerning this bizarre fibre-tract in this memoir which cannot be summarised. There is also an excellent bibliography. G. ELLIOT SMITH.

## **PATHOLOGY.**

**NEW RESEARCHES UPON THE NEUROFIBRILS.** G. MARINESCO, (369) *Rev. Neurol.*, 1904, No. 15, p. 813.

RAMON Y CAJAL has recently described certain characteristic changes in the spinal and cortical nerve cells in cases of experimental rabies in dogs. These changes, observed in preparations by his new silver method, consist mainly in simplification of the fibrillar network, fusiform thickening of the primary fibrils and the appearance of abundant yellow granules in the nucleus. Marinesco has already, in a previous paper, confirmed these observations, and now records the results of some further investigations into the

characters of the special nerve cell lesions of rabies. He has examined the cord and medulla of several rabbits which had been injected with the virus of this disease.

Swelling of the neurofibrils occurs in the nerve cells containing black fibrils as well as in those having red fibrils. In the latter the swollen fibrils, in their shape and granular appearance, simulate the Nissl-bodies. In the cells with black fibrils, the swellings in the course of the fibrils occur only at the periphery of the cell. The nuclear and nucleolar lesions are especially noteworthy. Normally in these silver preparations the nucleolus appears as if composed of a large number of reddish brown uniform granules. In rabies these granules are diminished in number and are unequal in volume. At the same time there are scattered throughout the nucleus from one to twenty or more generally larger corpuscles of various forms and sizes, and having a reddish yellow colour. In the interior of these corpuscles one or two granules having the same characters as the nucleolar granules may often be observed. In the spinal ganglia the large cells generally show loss of the fibrillar reticulum in the central part; the cells with concentric fibrils, or the clear cells, are affected to a less degree. The cells of the capsule show evidence of recent proliferation.

Marinesco considers that the thickening of the fibrils and the nuclear changes described are almost specific lesions of rabies. He has not found them in any other pathological condition. He believes that the bodies of Negri are formed by an alteration in the cellular protoplasm. They are not found exclusively in rabies.

The author next describes the nerve-cell lesions in the spinal cords of three guinea-pigs killed by injection of tetanus toxin. They affect especially the cells with red fibrils; those with black fibrils are for the most part normal. The morbid changes range from simple granular disintegration and fragmentation of the fibrils to complete degeneration and extreme pallor of the cells. The fibrillar lesions may be diffuse or peripheral, or they may affect specially the region of the axis-cylinder. The nucleolus is pale and the number of its granules diminished. The intracellular canals are dilated.

He has also studied the cadaveric changes affecting the neurofibrils. It appears that at ordinary temperatures the fibrils begin to show granular disintegration within twenty-four hours. After this period the disintegrative changes are rapidly progressive. The cells with red fibrils are affected more rapidly than those with black fibrils.

In two cases of general paralysis he has observed thickening and granular disintegration of the neurofibrils of the cortical nerve cells. The various researches that he has made lead him to conclude that the nerve cells with red neurofibrils are much more



vulnerable than those having black neurofibrils. The secondary fibrils are also more easily injured than the primary ones.

Lastly, he refers to the question of the repair of neurofibrils, and asserts that there is evidence that the fibrils after undergoing various morbid changes may gradually regain their former structural characters. The limit of reparability is, however, easily overstepped. Once destroyed they cannot be formed anew. Why the neurofibrils of the cytoplasm should thus be incapable of regeneration, whilst those of the peripheral nerves can exhibit this phenomenon, is a question that it is difficult to answer in the present position of knowledge.

W. FORD ROBERTSON.

**CHANGES IN THE SPINAL CORD OF THE TADPOLE CON-(370) SEQUITIVE TO AMPUTATION OF THE LIMBS.** BRISSAUD and BAUER, *Rev. Neurolog.*, Sept. 15, 1904, p. 929.

IN this paper, read at the Congress of Alienists and Neurologists in Pau (Aug. 1904), the authors continue their researches on the changes in the spinal cord of tadpoles as the result of experimental lesions. The specimens were all hardened in Zenker's fluid and stained with Nissl's method.

If two tadpoles of the same age undergo on the same day the same amputation, and should the severed limb in one case become replaced, in the other not, examination of the cords later will reveal a marked difference between the two lumbar enlargements.

In cases where replacement of the limb occurs, one may observe the progressive nature of the medullary repair process. It is never, however, complete; that is to say, though the regeneration of the limb be complete, there is no corresponding *restitutio ad integrum* in the cord. The motor cells most severely altered become the victims of phagocytes; a real neuronophagy (which may be bilateral) was frequently observed. Apparently simple nerve-cells in the neighbourhood are capable of adapting themselves to altered conditions and taking on the functions of the destroyed large cells. No karyokinesis was noted.

S. A. KINNIE WILSON.

**THE UNION OF DIFFERENT KINDS OF NERVE FIBRES.** J. N. (371) LANGLEY and H. K. ANDERSON, *Journ. of Physiol.*, Vol. xxxi. p. 365.

THE work done by previous observers on the subject is reviewed and their experiments repeated and extended by the authors of this paper. Cats and rabbits were employed for the purpose. Union of somatic nerves is first considered. The authors agree

with other observers that when two somatic nerves are divided and their central ends sutured together, no union of the two nerves takes place. If the peripheral ends of two divided nerves are sutured together there is no union of the two. In some of the experiments there appeared to be union when one nerve was stimulated by a faradic current, but it was found to be due to the presence of new fibres which had grown into the nerves from the central ends. After the central ends were severed, contraction still took place on stimulation; this the authors consider to be an axon reflex. It did not occur where there had been no reunion with the central ends. Bethe, on the contrary, attributes it to a direct union of the peripheral fibres, a conclusion with which Langley and Anderson disagree.

Somatic efferent fibres are found to unite with pre-ganglionic fibres, thus, after suture of the central end of the fifth cervical nerve with the peripheral end of the cervical sympathetic, faradic stimulation of the fifth produced the usual effects of the sympathetic on the eye. The same results were obtained when the phrenic nerve was united with the cervical sympathetic.

Pre-ganglionic nerves are also able to form a union with efferent somatic nerves; the central end of the cervical sympathetic was made to unite with the peripheral end of the divided recurrent laryngeal nerve, and in other experiments with the spinal accessory and phrenic. The authors believe that small nerve fibres can much more readily unite with large ones than large ones with small.

Experiments on the union of somatic afferent nerves with pre-ganglionic nerves were conclusive in proving that these fibres do not unite. Where the peripheral end of the cervical sympathetic was sewn into the skin of the neck, it was found seventy-nine days afterwards that numerous afferent fibres had grown into the nerve for variable distances, but had formed no functional connection with the superior cervical ganglion.

Pre-ganglionic fibres are incapable of forming a functional union with post-ganglionic fibres. Post-ganglionic fibres, too, are unable to unite with pre-ganglionic.

In one experiment on the union of post-ganglionic nerves with somatic efferent nerves, a negative result was obtained.

The authors are enabled from the above to draw some general conclusions:—

1. The central end of an efferent fibre can make functional connection with the peripheral end of any other efferent fibre of the same class whatever be the normal actions produced by the two nerve fibres.

2. The central end of any efferent somatic fibre can make functional union with the peripheral end of any pre-ganglionic

fibre, and the central end of any pre-ganglionic fibre can make functional connection with the peripheral end of any efferent somatic fibre.

3. Peripheral ends of cut nerve fibres exercise a chemiotactic influence on the central ends, varying in extent between different classes of fibres.

4. Nerve fibres with regard to possibility of union may be reduced to three kinds:—Efferent fibres proceeding from the spinal cord. Post-ganglionic nerve fibres. Afferent nerve fibres. Union is not possible between any of these three kinds.

PERCY T. HERRING.

**ON AUTO-GENETIC REGENERATION IN THE NERVES OF THE**  
(372) LIMBS. J. N. LANGLEY and H. K. ANDERSON, *Journ. of Physiol.*, Vol. xxxi., 1904, p. 418.

A NUMBER of experiments on young cats and rabbits are recorded with the view of confirming or disproving the autogenetic theory of nerve regeneration which has recently been so strongly advocated by Ballance and Purves Stewart, and by Bethe. Two questions are mainly dealt with: (1) whether nerve fibres which are permanently isolated from the spinal cord and spinal ganglia reform medullated sheaths; and (2) whether they acquire a motor action in skeletal muscle. In the first experiment, where the sciatic nerve had been divided in a kitten and the cut end sewn to the deep surface of the skin, stimulation of the internal popliteal one hundred and nineteen days later caused contraction of the gastrocnemius muscle, and stimulation of the external popliteal nerve was followed by contraction of the extensor group of muscles of the foot; neither nerve nor the sciatic where it joined the skin gave rise to any reflex body movement. No new fibres were found connecting the cut ends of the sciatic nerve. The authors admit that this experiment appears to support the autogenetic theory, but do not regard it as conclusive; the histological appearance was in favour of nerve fibres having grown in from other sources in the surrounding tissue. In another experiment of a similar nature it was definitely proved that a certain number of fibres had found their way from the central end of the sciatic into the peripheral part. Other experiments convinced the authors that the method of experiment was unsatisfactory, and that previous observers had fallen into the same error. It is a matter of extreme difficulty to prevent fibres from injured tissue from growing into the peripheral end of a divided nerve, and so establishing a connection between it and the central nervous system. In subsequent experiments the sciatic nerve was cut as

before ; some two hundred days or more later the sciatic nerve was again cut and other nerves which might have formed a connection also divided. Twelve days later the animal was killed and stimulation of the peripheral end of the sciatic now gave no results. Microscopically there were present a few degenerated medullated fibres which had evidently grown in and been severed from the central nervous system in the last operation. If autogenetic regeneration does occur, the number of fibres doing so is small and they had in every case formed a central connection, and underwent degeneration on section from it. The authors have good grounds for believing that the fibres found in the peripheral portion of a divided nerve have grown there from the central end of the same nerve or from some other nerve in the vicinity. They suggest that this establishment of a central connection by the divided nerve may have been the real reason for the positive results obtained by supporters of the autogenetic theory.

The authors conclude that their experiments, so far as they go, are decidedly against the autogenetic theory.

PERCY T. HERRING.

**BILATERAL LOSS OF POST-CENTRAL CORTEX, APPARENTLY  
(373) CONGENITAL, IN AN ADULT. H. M. TURNBULL, *Brain*,  
Summer 1904, p. 209.**

It is quite impossible in an abstract to do justice to the many interesting features in this case, which is elaborated clinically and pathologically, macroscopically and microscopically, in a most methodical and complete way. Two large cysts, which on examination were devoid of cells capable of exercising the functions of cortical tissue, were found to replace the occipital lobes, the cunei and precunei, practically all the temporal lobes and all the parietal lobes save the ascending parietal and anterior half of the supramarginal convolutions of a young adult aged 24, who had died from severe burns.

For the details of the examination in regard to optic nerves, tracts and radiations, grey nuclei and internal capsules, cortex and cyst, the reader must be referred to the original.

On many questions of dispute in central structure and physiology the case recorded sheds light. For instance, the discussions on the bundle of Türck, the corpus callosum, the retrolenticular portion of the internal capsule, the optic paths, and Gudden's tract may be specified.

Excellent photographs and drawings accompany the paper.

S. A. KINNIER WILSON.

**NOTE ON A CASE OF DEFECTIVE DEVELOPMENT OF THE  
(374) LATERAL CEREBELLAR LOBES IN A DOG. THOMAS  
LEWIS, *Brain*, Spring Number, 1904, p. 84.**

THIS is an account of the pathological findings in the case of a dog, whose only obvious clinical sign was circus movement to the left, a feature which had been observed ever since the dog came under notice four years previous to its death.

T. GRAINGER STEWART.

**STUDY OF THE RETINA IN TABETIC AMAUROSIS. LÉRI, *Nouv.  
(375) Icon. de la Salpêtrière*, July-Aug. 1904, p. 304.**

IN this paper Léri continues his research in the domain of tabetic amaurosis, the clinical part of which is succinctly yet very completely handled in his Thesis (Paris, 1904). The frequency of the condition is out of all proportion to the poverty of the anatomopathological work on the subject at present available.

Popoff in 1893, noticing that in a case of tabetic optic atrophy the second pair were very small in the neighbourhood of the eyeball, concluded that the morbid condition began in the cells of origin of the optic fibres, viz., those of the retina.

This retinal theory was supported by the few who have written on the subject till the appearance of the results of Holden (1899) and Moeli (1900).

It must, of course, be obvious that, as modern teaching holds the most distant prolongations of the neurone to be the first sufferers in a cellular lesion, we should, *a priori*, have expected the maximum of degeneration, not near the eyes, but in the neighbourhood of the primary optic centres, the external geniculate bodies, etc. Moeli and Holden entirely failed to discover any corroboration of this, as also has Léri, after the minute examination of the whole extent of the optic paths in eleven cases of tabetic amaurosis.

It is, then, not too much to consider as disproved the retinal theory of this condition. Tabetic atrophy does not begin in the retina, nor is it a primary degeneration, selecting the cells of origin of the optic nerves, viz., the multipolar cells of the retina.

The interesting and somewhat unexpected fact is noted, that in no case was there absolute absence of these ganglion cells, although the optic nerves were sometimes without a single sound myelinated fibre. Apparently the two conditions are independent one of the other. Other retinal changes are considered. There are excellent photo-micrographs.

S. A. KINNIER WILSON.

**THE PATHOLOGICAL ANATOMY OF ONE TYPE OF HEREDITARY CEREBELLAR ATAXY.** A. RYDEL, *Nouv. Icon. de la Salpêtrière*, T. xvii., No. 4, 1904, p. 289.

THIS work describes the clinical course and the pathological anatomy of a case of Marie's ataxy.

A detailed account is given of the symptomatology of the disease and morbid anatomy of the nervous system in a brother and a sister of Dr Rydel's patient, which have been published in 1900-1901 by Vincelet, and by A. Thomas and J. Roux. The clinical events in the case here reported were as follows:—The antecedents presented nothing remarkable. The illness commenced at the age of twenty-six years with weakness of the legs associated with tingling and cramp-like sensations. The lower limbs became easily fatigued. The patient gradually developed pains in the loins, and his gait became reeling. His speech became hesitating and indistinct, and attacks of complete loss of sight lasting a short time troubled him.

In 1891, three years after the onset, the condition was as follows:—Fixed expression, marked Rombergism, gait ataxic and lurching, no dragging of feet, lateral oscillation of fingers when spread, general muscular rigidity, choreiform twitchings in all the facial muscles, in the thenar muscles, and in the thighs and loins. Tremor of the tongue, tingling and cramp in the legs, pains in the loins.

Sensibility to touch and to pin pricks was lost over the feet and legs, much diminished over the hands and forearms.

Slight tactual loss over the face. Heat was everywhere recognised, cold was only felt upon the trunk.

Taste, smell and hearing were much diminished upon the left side. Marked contraction of both visual fields, and especially of the left field. Ocular fundi normal. Reaction of pupils to light absent. Reaction to accommodation very slow and feeble. Nystagmus.

Three years later he had developed a marked lateral curvature of the spine. The abolition of the light reflex was no longer present.

White matter. With the exception of the regions occupied by the pyramidal fibres, the white matter stained with great difficulty by the Weigert method, and was composed of fibres much smaller than normal.

In the posterior columns Lissauer's zone showed marked degeneration. Goll's column was markedly degenerate throughout the cord. The middle-root zone of Burdach's column was degenerate in the cervical region.

The direct cerebellar tract, Gowers' tract and the ventro-lateral regions were markedly degenerate. The direct cerebellar tract showed less and less degeneration from below upwards, till at the level of C<sub>4</sub> it presented no abnormality. Medulla.—The posterior longitudinal bundle, the internal arcuate fibres and the reticular formation showed marked degeneration. The central part of the restiform body was much affected. The olives were normal. The walls of the fourth ventricle were considerably thickened. The degeneration in Gowers' tract could be followed to the anterior limit of the pons.

The cerebellum was very small, the reduction being chiefly in the white substance. The cells of Purkinji and the nuclei were normal.

The author points out the similarity of the pathological anatomy in the three members of this family, and he considers these cases to be examples of a disease midway between Marie's ataxy and Friedreich's disease.

JAMES COLLIER.

## CLINICAL NEUROLOGY.

**THE PATHOGENESIS OF OBSTETRICAL PARALYSES.** THOYER-  
(377) ROZAT, *L'Obstétrique*, Sept. 1904.

It has been usual, for some years, to regard the elongation of the roots of the brachial plexus, and not their direct compression, as the cause of these paralyses. In cephalic presentations, after the head is born and there is difficulty with the shoulders, asynclitic tractions are made in order to get the anterior shoulder to emerge from under the pubic arch. In this way the roots of the brachial plexus, especially the fifth and sixth roots, are overstrained, and finally rupture. Paralysis of the superior type then results, which was formerly attributed to the direct compression of these two roots over Erb's supra-clavicular point. Similarly in breech cases, the mechanism necessary to free the arms of the fetus leads to a stretching of the branches of the plexus where they cross the capsule of the humerus.

The author observed a paralysis of the arm produced under conditions where elongation or indirect rupture of the roots of the plexus seemed impossible. There was difficulty in the birth of the shoulders, and the anterior one became fixed behind the symphysis. The posterior shoulder being more accessible its arm was disengaged and brought down. The anterior arm, however, was still the one paralysed, and this was probably to be explained from the fact that pressure had been exerted at the anterior shoulder upon Erb's point.

The author attempted to confirm this view of the production of such paralyes by experiments made on dead fœtuses, imitating as far as possible the actual conditions clinically present. He avoided making asynclitic tractions, but purposely made a certain amount of compression over Erb's region. He concludes: (1) That temporary paralysis may result from elongation of the fifth root, but the factor of pressure upon Erb's point, as a cause, cannot be eliminated. (2) That severe lesions of the roots of the brachial plexus are far more often caused by compression than by simple elongation, which appears, in most instances, to be only a predisposing factor. Even a moderate pressure on Erb's point is capable of damaging the upper roots of the plexus.

OLIPHANT NICHOLSON.

**VERTEBRAL DISEASE IN TABES DORSALIS.** FRANK, *Wiener* (378) *klin. Wochenschr.*, 25th Aug., p. 919.

THE writer records at great length and detail an undoubted case of locomotor ataxia in a man aged 55. No history of syphilis was obtainable, but there developed in a year shooting pains, difficulty in walking, diplopia, loss of pupil-reaction to light, incontinence of urine and fæces, and loss of deep reflexes. The point for which the case is recorded is the osteo-arthritic changes in the spinal column, consisting of enlargement and roughening of the spines of C. V. 7 and D. V. 1, scoliosis, and great thickening, roughening and ankylosis of L. V. 2 and 3. There is a skiagram of the condition in the lumbar vertebræ and a copious bibliography of the vertebral changes in tabetics.

JOHN D. COMRIE.

**SOME CASES OF FAMILY DISSEMINATED SCLEROSIS.** E. S. (379) REYNOLDS, *Brain*, Summer 1904, p. 163.

THE fact that family insular sclerosis is distinctly rare justifies the recording of the cases here detailed; cases, further, which illustrate the aberrant types of the disease often so difficult to diagnose with certainty. In the first series, the second, fourth and sixth children have disseminated sclerosis, the father is melancholic and probably suicidal. A first cousin (male) of his died from advanced disseminated sclerosis, and another cousin (female) suffers from marked exophthalmic goitre. The mother's sister suffers from insomnia and melancholia. The other children were three in number; the eldest died in an asylum from melancholia; the third is affected periodically with severe sciatica; the fifth is said to be well. The second series concerns two sisters. The author



epitomises the points in his cases illustrative of the aberrant types of the disease:—

Irregular subjective patches of numbness, often varying, disappearing and reappearing; sudden falls from giving way of a leg; slight paralysis of hands, lasting for months or years; transitory paralysis of eye muscles; optic atrophy; subjective feelings of giddiness; alteration of handwriting; sudden dropping of articles held in the hand; alteration of speech, even very slight; ataxic movements of hands and sometimes of feet; spastic condition of legs, with true ankle clonus; Babinski's toe extension reflex.

S. A. KINNIER WILSON.

**A CASE OF SO-CALLED IDIOPATHIC CHRONIC INTERNAL (380) HYDROCEPHALUS (IN THE ADULT) AND A CONTRIBUTION TO THE STUDY OF OBJECTIVE NOISES IN THE HEAD.** ALFRED FUCHS, *Arch. a. d. Neurol. Institut, Wien*, Bd. xi., 1904, p. 62.

THE condition of idiopathic chronic internal hydrocephalus in the adult is rare, and little is known about it clinically or pathologically: Fuchs cannot find in literature a single case in which a certain diagnosis was made *intra vitam*.

Full notes, clinical and pathological, are given of a case whose chief interest lies in the fact that the symptoms, more especially the characters of a murmur heard on auscultating the cranium, made the diagnosis of endocranial aneurism appear certain, whereas no lesion of the vessels was found at the autopsy. The following is a resumé of the notes of the case.

When first seen, the patient, a woman aged 30, gave a history of having suffered for three years from head pains, occipital in situation, often very violent for some hours or a day, with intermissions lasting days or weeks: for almost three years she had had noises in the ears and some "enlargement" of the eyes and slight goitre were noted. There had been no vomiting or giddiness; there was no history of lues or injury to head. On examination, the chief points noted were: pulse rate 130, no degeneration of vessel walls, fine tremors of outstretched hands, slight symmetrical enlargement of thyroid gland with no thrill or bruit, slight exophthalmos, not pulsating. Examination of the nervous system gave negative result with the exception of double optic neuritis (4 D. swelling R., 3 D. L.) and a murmur heard on auscultating the cranium; the murmur was of a blowing character, synchronous with the pulse, and could be heard over the whole cranium, but more loudly on the right side; it was loudest over the right mastoid process and temporal region, and it was heard with special clearness

through an otoscope in the right ear; compression of the right carotid artery made the murmur, subjective and objective, disappear.

A certain diagnosis was made of aneurism of the right carotid artery at the cerebral end of the carotid canal.

The patient refused to persevere with any treatment. No material change was noted for about five months, when she observed diminution of vision, especially of the right eye; three weeks later, attacks of giddiness appeared and, soon, vomiting when these attacks were at their height; she became bedridden, passing urine and stools involuntarily, and was compelled to lie on her right side because any other position caused violent giddiness. At this date, i.e. about seven months after the first examination, she came under observation again. She complained of intolerable head pains, especially occipital, and agonising noises in the head; she assumed a forced position on the right side, with trunk and legs fully flexed. Exhaustive examination was impossible, but there were no gross signs of paresis, anæsthesia, etc.; the left pupil was slightly greater than the right, there was a trace of nystagmus on extreme movement of the eyes, optic neuritis was intense; the systolic blowing murmur in the head had markedly increased in intensity and could be heard with the ear held at a distance of 1 to 2 mm. from the right temple; it had the same points of maximum intensity as before; compression of the right carotid artery did not make the murmur quite disappear and had no effect on the subjective giddiness. About two hours later, she seemed brighter and raised her head a little and spoke to her attendant; in the middle of a sentence she died quite suddenly.

The original diagnosis seemed to be confirmed by the later course of the case; the very sudden death was more difficult to explain.

*Autopsy.*—No lesion of the vessels was found. The convolutions of the brain were flattened; the lateral ventricles and more especially the third ventricle were markedly dilated and filled with clear, yellow fluid; the ependyma was finely granular, the plexus of the fourth ventricle thickened, and the foramen of Magendie patent. On microscopical examination, there were clear signs of leptomeningitis, especially in both parietal regions, and the cerebral cortex generally was hyperæmic. The plexuses showed at places slight small-cell infiltration. Nothing abnormal could be detected in the acoustic region of the base of the skull on either side; section of the left petrous bone was made and showed every part, including the carotid canal, intact; unfortunately, this was not done on the right side.

From lack of personal experience, Fuchs does not feel justified in attempting to explain the hydrocephalus; he draws special attention to the patency of the foramen of Magendie.

He finds that very few cases of idiopathic *chronic* hydrocephalus in the adult, with autopsy, are on record; acute cases—amongst which he includes Annuske's case, which is generally recognised as the first recorded case of the chronic variety—and cases occurring before the thirteenth or fourteenth year of life are excluded. Amongst the few genuine cases found, he gives special prominence to a case recorded by Oppenheim, because of its striking similarity to his own case in clinical course and symptoms; the diagnosis *intra vitam* was tumor cerebri, while the autopsy disclosed no tumour, but a condition of internal hydrocephalus very similar to that seen in Fuchs' case. Fuchs believes that not a few cases which have been recorded under a different diagnosis, *e.g.* endocranial aneurism and tumour, and in which the diagnosis was not confirmed by autopsy, are probably examples of chronic internal hydrocephalus; several such cases are quoted in considerable detail, showing a remarkable clinical similarity to his case.

The symptom most likely to cause difficulty in diagnosis is the murmur heard on auscultating the cranium, of a blowing character, synchronous with the pulse; the difficulty is all the greater, where the murmur is more or less localised in situation and clearly related to the blood flow through a particular vessel (*e.g.* a carotid artery). A murmur with these characters is generally recognised as a certain sign of endocranial aneurism, but it would appear that it may occur in hydrocephalus. Similar murmurs, synchronous with the pulse, are noted in other conditions:—

1. Tumours: (a) where the tumours are very vascular; (b) where the tumours cause pressure on surrounding vessels.

2. Anæmia and Graves' disease.

3. In children, a systolic murmur is well known.

A bruit heard more or less equally well over the whole skull may make one hesitate to diagnose aneurism, in which the bruit generally preponderates on the side on which the aneurism occurs.

There is no clear explanation of the murmur heard in hydrocephalus. On the analogy of other murmurs, it is most probably due to a diffuse relative narrowing of the vessels, caused by the increased brain tension. Why, however, the maximum murmur should be so strictly localised at one spot, usually the region of the ear, does not appear. Fuchs suggests that an important part may be played by pathological changes—caused by increased intracranial pressure—in the sound-conducting power of the skull and its contents.

In conclusion, two points in the symptomatology are mentioned, which may help towards a diagnosis between aneurism and the form of hydrocephalus considered:—

1. Optic neuritis is generally absent in aneurism.

2. The two symptoms, head pains and noises in the ears,

appear about the same time in very many cases of aneurism ; while, in the recorded cases of hydrocephalus, the head pains have almost always appeared a long time, usually years, before the noises in the ears.

A. W. MACKINTOSH.

**ON FAMILY SPASTIC PARAPLEGIA.** L. NEWMARK, *Deutsche* (381) *Ztschr. f. Nervenheilk.*, Bd. xxvii. H. 1 u. 2, 1904, p. 1.

IN the year 1893 the author described two families, several members of which suffered from spastic paraplegia. Owing to the death from phthisis of one of the members of the second family, an opportunity has been afforded of examining the nervous system. In this family there were fourteen children. The parents had been healthy individuals, but the mother had died suddenly. Of the children, four had died in infancy and one of tuberculous meningitis. Of the nine living children, seven were affected—five boys and two girls.

The third child first showed signs of the disease when eight years old ; death occurred from phthisis when twenty-two years old. In only one member of the family was there any alteration of sensation, and in none was there any disturbance of bladder function, nystagmus, strabismus, changes in the pupil, fundus, or mental impairment. The history of this third child was as follows:—

He was said to have been well till eight years of age, when he had "typho-malarial fever." The peculiarity of his walk had been noticed before the illness, but after recovery his gait was worse.

The hip and knee were flexed and adducted, and there was double equinus. The legs could be put straight by considerable force. The knee-jerk was exaggerated ; there was slight ankle clonus, and all the superficial reflexes were very active. The functions of the upper extremities were unimpaired, but all the reflexes were increased. The patient was able, in spite of this spastic condition of the legs, to walk long distances without fatigue.

The pathological examination revealed in the sacral region of the cord a slight affection of the pyramidal tracts, but the anterior and posterior roots and the posterior column of the cord appeared normal.

In sections through the middle of the lumbar region there was, in addition to the change in the pyramidal tracts, a change in the posterior columns involving the root zone of Flechsig.

In sections stained by V. Giesson's method the vacuolation of the area affected was very striking.

In the upper lumbar region the degeneration of the pyramidal tracts and of the posterior column was about the same as in the

section through the lower lumbar region. The cells of Clarke's column were fewer in number than normal, but the cell in the anterior horns appeared normal.

In the thoracic region the affection of the pyramidal tracts and posterior column was still present, but the number of cells in Clarke's column was more numerous.

In the cervical and upper cervical region no change was visible in the pyramidal tracts, but the posterior median column of Goll showed marked sclerosis, and the sclerosis could be traced up to the cells of the nucleus of Goll's column.

The anterior and posterior roots were normal.

The author compares the pathological examination of this case with those of Strümpell and Bischoff.

Degeneration of the pyramidal tract and of the posterior column has been present in all five recorded cases in a variable degree. The direct cerebellar tract was markedly involved in Strümpell's case, while the cells of Clarke's column were only affected to any extent in the present case.

The degeneration of the posterior columns with its vacuolated appearance is compared to that which is found in subacute combined degeneration of the cord, and the pathological similarity between the two conditions is pointed out.

When the clinical and anatomical features of the case are taken into consideration, it seems probable that the affection of the pyramidal tracts and Goll's column is of endogenous origin and dependent on some congenital failure.

The paper is illustrated by some excellent photographs, which demonstrate the grosser morbid changes most clearly.

F. E. BATTEN.

#### **PATHOLOGICAL SLEEP IN CASES OF CEREBRAL TUMOUR.**

(382) FRANCESCHI, *Rivista di Patolog. nerv. e ment.*, Ottobre 1904, p. 457.

THE difficulty is to decide exactly in such cases between dulness, somnolence, stupor, or even coma, and sleep, to which the epithet pathological is applied only as regards its length and uninterruptedness. In the case under consideration a young woman of 26, apparently healthy, commenced to be oppressed by an irresistible desire for sleep during the day—later, headache, giddiness, and occasional vomiting accompanied the drowsiness, which increased in intensity up to the end. All the time, be it noted, the patient could be roused when spoken to, and replied to questions with unflinching coherence. Post-mortem, a tumour was found occupying the opto-peduncular region, extending to the anterior margin of the

- pons, and compressing and distorting the arteries in the circle of Willis. The optic nerves and the left optic bandette were atrophied. Theories to account for the pathological amount of sleep are advanced and criticised. It was obviously not due to increased intracranial tension. The view that involvement of the third ventricle and hypophysis by any lesion, including a neoplasm, is associated with alteration in sleep, is not accepted, except in so far as such lesion may impair circulation in the basal arteries. This is the hypothesis considered most feasible by the author—viz., cerebral anæmia, due to interference with the arteries of the circle of Willis.

S. A. KINNICK WILSON.

**A CASE OF SYPHILIS OF THE NERVOUS SYSTEM PRESENT-  
(383) ING CLINICALLY AN AMNESTIC SYMPTOM COMPLEX  
WITH AUTOPSY. EMMA W. MOOERS, *Am. Journ. Insan.*,  
July, 1904, p. 11.**

THIS article is a description of the symptoms and physical signs and the microscopical changes found post-mortem in the nervous system. The case is that of a man who, in 1883, at the age of twenty-four, contracted syphilis. In 1894 he had a severe convulsion of unknown cause, leaving no after effects. He first showed symptoms in 1892, when he became nervous, easily tired, and lost appetite. In 1899 his gait became affected; when made to walk a straight line he deviated generally to the left; there was no ataxia or Rombergism; he had tremors of his fingers and tongue, the right angle of his mouth was a little lower than his left, and the right naso-labial fold was flattened; mentally he showed a mild euphoria and a certain easy-going unconcern, otherwise he was normal. In April 1900 nystagmus was noted. In December 1899 he had a convulsion with movements of the left arm and leg, but he was quite recovered by the evening. In June 1900 he had three convulsions following one another rapidly, in which his whole body became stiff; in the last the right side of his face was more relaxed than his left; his head and eyes were turned to the left; his right arm and leg were stiffer than his left; he was unconscious till next day. Next day he was dull, sleepy, and had completely lost his memory for recent events, which he never regained, he could, however, remember events of long ago; left hemianopia was found, which, however, only lasted for two days; he also saw red ants about his bed, red letters about the room, and the ice which was given him he said was red. Mentally he was now dull but had not lost his personality; speech became gradually thicker, and in November 1900 was noticeably so. In December 1900 he had some

difficulty in starting to urinate, but there was never any retention; he had occasional attacks of violent vomiting. On January 26 he had about seventy convulsions, his head and eyes were turned to the right, and his right side was especially affected; he remained unconscious all night, and died next morning.

*The autopsy.*—There was general thickening of the membranes of the brain, especially at the base of the brain and in the spinal cord; slight bulging of the right side of the pons was noted; nothing was found in any other organs.

*Microscopical.*—The membranes were infiltrated in the fissures and deep sulci of the brain with various forms of lymphoid cells, also the blood-vessels, some showing typical endarteritis obliterans. There were present in the membranes and in the brain tissue numerous focal lesions; these consisted of masses of cells enclosing foci of necrotic material, the cells being those of granulation and lymphoid tissue. Interspersed among the cells were numerous capillaries, and the necrotic focus was surrounded by elongated cells. Giant cells were sometimes present. The adjacent brain tissue showed an increase of neuroglia cells, some attaining huge size; the location of these foci in the nervous system was in the right hemisphere, the vessels and pia of the anterior perforated spot, the temporal lobe, the cornu ammonis and peduncle, the thalamus, the pulvinar, the red nucleus and splenium of the corpus callosum, the hippocampal gyrus; in the left hemisphere the peduncle, frontal and temporal lobes, hippocampal gyrus, fissure of the occipital lobe; the optic chiasm and optic nerves. The glia cells at the surface of cortex were thickened at places, and also between the cortex and white matter; the cells of the cortex had undergone typical "fever" alteration; in the pons the bulging was found to be due to a large focal lesion, 6-10 mm.; the pyramids were both somewhat degenerated. In the cord the arteries were affected, and the membranes like those of the brain. In the lumbar region the ventral nerve roots were markedly degenerated; here also a peculiar glassy hyaline tissue was found replacing the nerve tissues. There was marked marginal sclerosis at all levels; there was a small neoplasm on the right third thoracic dorsal root similar to the foci of the brain, only more vascular; secondary degeneration of the pyramidal tract was found on both sides.

The following are some of the correlations between the individual symptoms and anatomical lesions: The alteration of the gait due to the foci in the left peduncle and right side of the pons; the bladder symptom to lesion found in sacral cord; the transient left-sided hemianopia to involvement of the right pulvinar (the correlation of the mental symptoms is not attempted). A special feature drawn attention to at the end of this article is the importance of the diffuse degeneration and glia increase in the brain cortex, which

is not dependent on any of the focal lesions, and which has frequently been found in the cord but rarely in the brain.

J. C. MOTTRAM.

**THE PRE-EPILEPTIC STAGE AND ITS IMPORTANCE FOR**  
**(384) PROPHYLAXIS.** L. J. J. MUSKENS, *Nederl. Tydschrift voor*  
*Geneeskunde*, 1904, d. 1, 1.

DEVELOPMENTAL studies on epilepsy regard either the ontogenetic development of the disease, i.e. in the individual person, or its phylogenetic development, i.e. the way in which the disease in past generations came into existence and gradually arrived at its present features in the human subject. Just as morphology benefited largely from the combined study of onto- and phylogenesis, so we may expect pathological results from following the same method. In former publications the author has shown that the epileptic discharge is rarely or never a sudden accident, but that it is always preceded by more or less striking abnormal symptoms of psychical, sensory, motor or vasomotor character. In the same way the outbreak of genuine epilepsy in young persons is nearly always preceded by a stage of latent epilepsy. This stage may commence even in the intra-uterine period. As to the psychical symptoms, they are, it appears, less constant, but if present are not dependent on what even may be in a given case the organic cause of the disease. Slowness of reaction, backwardness in learning, but more often lack of memory with sufficient mental capacity, tendency to vehement outbreak of temper. Very remarkable is a peculiar apathy met with in these children. The mothers will tell that the children rarely or never weep or display emotion if brought into the presence of a dead body. It is very difficult in many cases after definite epilepsy has declared itself, to estimate the psychical condition, because after the treatment with bromides, we have to deal with the difficulty of ascertaining whether the defective brain power is due to the epileptic constitution itself or to the sometimes large doses of drugs which have been administered.

More frequent and less easy to examine are the abnormal conditions of sensibility to pain which can be found in the pre-epileptic state. The mothers often remark on the striking circumstance that the children in question appear to disregard the drawing of a tooth, a whitlow, or an extensive burn. Not uncommonly it is observed that this insensibility to pain is at one time more pronounced than at another. This peculiar insensibility has nothing to do with the mental condition, because the most striking examples are related about very intelligent patients and



in contrast to the psychical and motor disturbances. Those of cutaneous sensibility can be regarded as of the same order in the pre-epileptic stage as those after the disease is fully developed.

Thirdly, the minor motor disturbances of the pre-epileptic stage are sometimes so clear, that regular anti-epileptic treatment may be indicated long before regular fits have presented themselves. Spontaneous convulsive movements during sleep and especially on rising in the morning are characteristic. In girls long before menstruation has appeared, very often at regular periods, these movements may become very troublesome, not uncommonly the first fit coming on at one of these periods which coincides with the first menstruation. The moments of waking in the morning and rising from bed must be regarded as predisposing moments of pre-epileptic convulsiveness.

AUTHOR'S ABSTRACT.

**STATUS EPILEPTICUS: A CLINICAL AND PATHOLOGICAL (385) STUDY IN EPILEPSY.** L. PIERCE CLARK and THOMAS P. PROUT, *Am. Journ. Insan.*, July 1904, p. 81.

THIS paper represents the result of the study of 38 cases of status epilepticus, and also of the pathological investigation of several cases of status and epilepsy.

It is impossible to give in any detail many of the interesting details which the authors have noted. Their clinical notes are full of valuable observations, and are well worth careful study.

The second part of the paper contains a brief account of the various views which have been held in regard to the pathology of epilepsy. They mention especially the work of Prus, who, as the result of many experiments, was of opinion that (1) the transmission of the impulses in epilepsy is through the extra-pyramidal tracts which transmit motor reflex impulses; (2) the sensory elements of the cortex must be irritated in order to produce the fit; (3) the fit appears to be a complicated reflex phenomenon.

The gross anatomical lesions found in status vary from slight stigmata of degeneration to positive deformity of the cranium or its contents.

Their microscopical researches were confined to the cortex. They employed the Nissl method for the study of the nerve cell, and the Robertson and Weigert methods for the investigation of the neuroglia. They examined the brains of 7 cases of status and of 12 cases of epilepsy.

They sum up the result of their findings thus:—

1. The first change appears in the nucleus of the cell first and most seriously involved. The nuclear membrane and the karyoplasmic network disappear and the karyoplasm becomes granular.

As a result the nucleolus becomes free, and in the process of making the section it is often abstracted outside the cell.

2. The granules of the chromatic substance disappear from the cell body leaving a dimly outlined cell framework, all trace of the nucleus disappearing, and only a shapeless mass of vacuolated protoplasm represents the cell.

3. The cortex becomes invaded with leucocytes, mostly large mononuclear and phagocytic in action.

4. The neuroglia proliferates to take the place of the destroyed nerve cells, and this is most marked in the outer cortical layer and occurs in proportion to the duration and severity of the epilepsy.

5. The destructive process involves chiefly cells in the second, and, to a less extent, in the third layers of the cerebral cortex.

They drew the following conclusions from their results:—

1. If the special involvement of any particular type of cell is indicative of the essentially sensory or motor character of epilepsy, it would seem that Prus was correct in concluding that epilepsy is essentially a sensory phenomenon as the cells involved more especially are those of the second and third layers.

2. The essential lesion of epilepsy pertains to the nucleus of certain of the cortical cells, and is of such a nature as to seriously jeopardise the cell for considerable periods and ultimately cause its destruction.

3. The chromatolysis in epilepsy is a nutritional change brought about by the nuclear toxin, since the nucleus presides over the process of elimination, absorption and digestion of the cell unit.

4. The rôle of the leucocyte is phagocytic.

5. The neuroglia overgrowth in epilepsy is one of its more remote sequences and probably occurs in response to toxic irritation.

They do not throw any new light on the pathogenic agents which give rise to the onset of the disease. The third part of the paper discusses the treatment of status epilepticus—sedative in the convulsive stage and stimulant in the stuporose stage. They specially warn practitioners against the sudden withdrawal of sedative treatment in any case of epilepsy, such being often the immediate cause of the onset of status.

The paper contains several good clinical charts showing the temperature, pulse and respiratory curves observed in status, and also some drawings and micro-photographs of their pathological findings.

T. GRAINGER STEWART.

**MYASTHENIA GRAVIS.** HENRY HUN, with Report of an Autopsy (386) by GEORGE BLUMER and GEORGE L. STREATER, *Albany Medical Annals*, Vol. xxv., No. 1, 1904, p. 28.

THIS paper contains a very complete clinical and post-mortem record of a case of myasthenia gravis, an excellent succinct resumé of the literature of the subject, and a good up-to-date bibliography.

The patient was a locomotive engineer, who died of the disease at the age of thirty-three, the symptoms having been present for two years. When first seen, a year before his death, all the muscles were weak, although there was no complete paralysis. A left-sided ptosis of gradual development had been the first evidence of disease. The symptoms, which were typical, presented several points of interest. Faradic stimulation of the median nerve produced increasing feebleness of contraction in the muscles supplied by it until the contraction ultimately ceased altogether. A rapidly-interrupted galvanic current applied to the muscles innervated by the median nerve was not followed by a diminution in the activity of their contraction. The red corpuscles numbered 5,900,000. Galvanism applied to the muscles and strychnine hypodermically were the only therapeutic measures which appeared to produce any beneficial effect. Slight exposure to cold made the patient much weaker. Towards the end he suffered from severe attacks of dyspnoea, in one of which he died.

A post-mortem examination was made seventeen hours after death, weather cold. The heart was very flabby, the lower lobes of the lungs markedly congested, and the spleen much reduced in consistency. No changes were found in the central or peripheral nerves, which were very carefully examined. The muscles were infiltrated with lymphoid tissue. The thymus gland showed similar changes, and there was a proliferation of its glandular elements. "The changes in this gland suggesting lymphosarcoma." No distinctive changes were met with in any of the other organs.

The results of previous autopsies are tabulated, and are as follows:—

Fatal cases, fifty; no autopsy in eighteen, an autopsy in thirty-two cases, negative findings in the nervous system in seventeen cases.

(a) Old hæmorrhage in corpus callosum, Berkeley, one case.

(b) Minute recent hæmorrhages, with unaltered red corpuscles, in floor of fourth ventricle and of aqueductus Sylvii, Hoppe, Charcot-Marinesco, Murri, Cohn, four cases.

(c) Slight changes in motor nerve nuclei in medulla, etc.

- (x) Chromatolysis, Widal-Marinesco, Murri, Burr and M'Carthy, three cases.
  - (y) Doubtful degenerative changes, Sossedorf, Myers, Batten, and Fletcher, two cases.
  - (d) Slight changes in cranial motor nerves.
    - (x) Smallness of fibres, Eisenlohr, Oppenheim, two cases.
    - (y) Exudate between fibres, Oppenheim, Fayerstajn, Liepman, three cases.
  - (e) Developmental abnormalities with normal nerve cells and fibres.
    - (x) Septum in aqueductus Sylvii with reduplication of the canal, Oppenheim, one case.
    - (y) Septum in central canal of cord with reduplication of the canal, Senator, one case.
  - (f) Sclerosis of spinal cord, Long and Wilkie, one case.
- Findings outside the nervous system :—
- (a) Multiple tumours, Senator, one case.
  - (b) Enlarged thymus, Hoedelmoser, Link, two cases.
  - (c) Lymphosarcoma of thymus, Weigert, one case.
  - (d) Lymphosarcoma of lung, Goldflam, one case.
  - (e) Lymphoid infiltration of muscles, Weigert, Goldflam, Link, three cases.

EDWIN BRAMWELL.

**MYASTHENIA GRAVIS.** GEORGE E. RENNIE, *Australian Med. Gaz.*, (387) May 1904.

DURING the last four years the number of cases of myasthenia gravis which have been reported has rapidly increased. The author in this paper contributes to the literature of this disease the clinical records of two cases which have come under his observation in Australia. The first case is an example of the less severe type of the disease, while the second, which terminated fatally, illustrates well the more acute form—the patient dying from respiratory failure. The author unfortunately was unable to obtain a post-mortem examination. The reports of both cases are most clearly and fully stated, and show well the clinical entity of the condition.

T. GRAINGER STEWART.

**CONTRIBUTION TO THE STUDY OF MYASTHENIC PARALYSIS.** (388) HYACINTHE DE LEON, *Nouv. Icon. de la Salpêtrière*, T. xvii, 1904, p. 269.

THIS article contains a very full report of a case of myasthenia gravis, which commenced in a peculiar manner, with hemicrania, vomiting, and external ocular paralysis. The patient has been

under observation for over nine years, and the clinical account is full of interest, as the progress of the disease has been carefully noted. Despite several severe attacks of respiratory asthenia the patient was still alive at the time this paper was published.

T. GRAINGER STEWART.

**MYASTHENIA PSEUDO-PARALYTICA (ERB'S DISEASE), WITH (389) ATROPHY OF THE TONGUE AFTER OVER-EXERTION.**

P. K. PEL, *Berlin. klin. Wchnschr.*, 29th August 1904, p. 917.

THIS case presents the well-known features of the disease, with the peculiarity that there was distinct atrophy of the tongue, which was unassociated with fibrillary tremor. Atrophy of the tongue has been present in several cases described by various writers.

Excessive exertion—the patient was a milliner who worked sixteen hours a day—was in the opinion of the author a factor of etiological moment in the case. On the death of the patient, which occurred during a breathless attack, no macroscopic changes were to be seen in the nervous system or muscles, but at the time this paper was written a microscopic examination had not been made. There were no signs of pneumonia. No mention is made of the condition of the thymus gland.

EDWIN BRAMWELL.

**ADIPOSI DOLOROSA (DERCUM'S DISEASE). DELUCQ and ALAUX, (390) *Presse Méd.*, Sept. 17, 1904, p. 594.**

THE writers state that since this disease was described by Dercum in 1888, there have been forty-two further observations. The present case of a woman, aged 61, still alive, of alcoholic parentage but healthy childhood, began at the age of 10 years shortly after a severe fall on the back of the head. In this fall she lost consciousness, though the senses soon returned. Shortly afterwards the eyesight began to fail and the left eye to be protruded, till at 20 blindness was complete. Since the age of 14 also there has been a great swelling accompanied by pain on the left side of the nose, and the left orbit is felt to be filled up in part by a bony mass. At the age of 30 a deposit of adipose tissue, accompanied by severe lancinating pains of paroxysmal character, appeared on the right leg, and both spread to other parts. The writer gives an elaborate description of the manner in which the fat is disposed (the parts chiefly affected being the neck, shoulders, upper arms, thighs and legs), together with a picture of the lower limbs. Considerable asthenia has also appeared, but there is no psychic change.

As to diagnosis, the writer makes no doubt, since the case presents the three cardinal symptoms of Dercum's disease, viz., irregular adiposity, great pain and tenderness, and asthenia. The psychic changes, which are by many regarded as another important symptom, and which are wanting in this case, he holds of little importance. He refers the cause to a probable bony orbital tumour of old standing, resulting possibly from the fall upon the head in early life, and pressing upon the pituitary body.

JOHN D. COMRIE.

**PSEUDOTETANY, AND TRANSITION FORMS BETWEEN TRUE (391) AND HYSTERICAL TETANY.** H. CURSCHMANN, *Berl. klin. Wchschr.*, Sept. 19 u. 26, 1904, S. 997 u. 1031.

THE author here discusses the occasional difficulty in differential diagnosis between true and false tetany, and suggests the possibility of the co-existence of both, or at all events of transitional types between them in one patient. He gives the details of two cases, the first being that of a man, aged 33, which he believes to be one of hysteria occurring in the male, only showing in the form of an attack of pseudotetany, in which the fundamental symptoms of genuine tetany (*e.g.* those described by Trousseau, Chvostek, Erb, etc.) were absent. He also cites another similar case in his experience, and then describes in case 2 (a female patient, aged 30, suffering from hysteria) a transition form between pseudo- and true tetany, in which tetanoid contractions occurred in the hands, which, together with the other symptoms observed, at first seemed to be hysterical in origin, but in addition to these the symptoms of Erb and Chvostek were also present, though that of Trousseau could not be elicited. He therefore concludes that in such cases we must either have a mixture or combination of the symptoms of hysteria with those of true tetany, or we must admit that what has hitherto been regarded as true tetany may originate in a purely hysterical condition.

In an addendum (p. 1033) he gives a short description of another case, that of a girl, aged 15, intermediate in character between cases 1 and 2.

W. E. CARNEGIE DICKSON.

**THE NERVOUS COMPLICATIONS AND SEQUELÆ OF WHOOP-(392) ING-COUGH.** NEURATH, *Arch. a. d. Neurol. Instit. Wien*, Bd. xi, 1904.

A LARGE part of this paper is devoted to an exhaustive critical survey of the literature of the subject, and will be found a valuable

source of reference by other workers in the same field. Convulsions, meningitis, cerebral palsy (hemiplegia, diplegia, and more complicated forms of paralysis, among others, cases simulating multiple sclerosis and Friedreich's ataxia), psychical disturbances, affections of the sense organs, cord lesions, and polyneuritis come within the purview of the writer, who has collected, abstracted, and arranged cases from all sources with a diligence which compels admiration. Sudden onset of cerebral complications points to intracranial hæmorrhage. In the early years of life meningeal hæmorrhage is more common than cerebral; the former causes irritative symptoms, while the latter produces paralysis. Rapid course with coma and convulsions is a sign of meningeal hæmorrhage. Hæmorrhage into the cord or medulla may occur. The raised intravenous pressure is less to blame for the hæmorrhages than a post-infectious change in the vessel walls. Toxic vascular lesions may be caused by whooping-cough itself, or by a previous or concomitant infection—influenza, measles, tuberculosis, pneumonia, etc. It is doubtful whether embolism or thrombosis ever occur as a cause of the cerebral complications of pertussis. The post-mortem findings in this condition have neither demonstrated any common anatomical basis nor have they shown any constant parallelism to the clinical features of the cases. Trephining and lumbar puncture have proved of no value in assuring the diagnosis of hæmorrhage. Such being the results of his inquiry into the literature, Neurath proceeded to verify or disprove them by a systematic search, histological and bacteriological, for definite lesions in a series of fatal cases of pertussis, chosen at random, some of them dying with nervous complications, others without these. In all, he examined twenty-five cases, and in fifteen found alterations in the leptomeninges—cellular infiltration, oedema, hyperæmia, hæmorrhage, and exudation. In other words, there was evidence of simple meningitis, bearing considerable resemblance to the meningitis serosa described by Quincke. A somewhat similar change has also been described in a few cases of other forms of infectious disease—typhoid, pneumonia, etc. Neurath's bacteriological examinations proving negative, he infers that the lesion is toxic. Of the fifteen cases giving positive results, twelve had had nervous symptoms, while only two of the ten in which the meninges were normal had suffered.

J. S. FOWLER.

**TWO CASES OF NERVOUS LEPROSY.** RAYMOND, *Arch. de* (1933) *Neurol.*, Aug. 1904, p. 97.

RAYMOND describes, in exhaustive detail, two cases of typical nervous leprosy, one from New Caledonia, the other from Indo-China. The clinical picture of the two was more or less the

same: itching of the nostrils, numbness in the extremities, followed by paralysis of certain muscular groups; diminution in the volume of the extremity; simian hand; red-brown patchy coloration of the skin in the affected area; loss of all sensation; loss of the stereognostic sense; marked thickening of nerve trunks, some of which were irregularly nodulated or moniliform; diminution of electrical reactions; considerable trophic disturbance in the shape of dry skin, bullæ and old cicatrices on fingers or toes; loss of tendon reflexes, etc.

He proceeds to give a differential diagnosis from syringomyelia, interstitial hypertrophic neuritis, and Aran-Duchenne muscular atrophy. As for Morvan's disease, the author holds very logically that his two patients were suffering from the syndrome or symptom-complex of Morvan (paresis, analgesia, trophic disturbances), but that it is common to several conditions, including some forms of syringomyelia, and some forms of leprosy.

The pathogeny of the disease is discussed, as well as several questions arising therefrom. He believes that the neuritis may, by direct spread, become intramedullary, and affect the posterior columns, as has been described by several authors. Déjérine, on the other hand, holds that a similar lesion in the posterior columns is common to all cachexias.

S. A. KINNIER WILSON.

**ON VIBRATORY SENSIBILITY.** MARINESCO, *Presse Méd.*, Aug. (394) 13, 1904, p. 513.

THE renewal of interest in the question of pallæsthesia is amply evidenced by the recent contributions of Minor and of Goldscheider in Germany, of Egger, Rydel in France and now of Marinesco. The latter gives a brief but complete historical resumé, and proceeds to analyse the results obtained from the examination of all the patients in his *service*. Egger's tuning-fork he found most satisfactory, although no adequate reasons are given for the choice of the instrument. No reference is made to the ingenious device employed by Rydel and Seiffert to overcome the difficulty of obtaining identical stimuli with the fork. Marinesco's own method is, after striking, to hold it out at arm's length until no audible sound impinges on the ear, then to apply it. This he calls his minimal stimulus. In the reviewer's opinion the method is thoroughly unsatisfactory, especially when others have been so successfully employed. The author apparently is conscious of the defects of his technique, for he puts in a plea for a standardised instrument.

As far as clinical results go, Marinesco fully admits the value of the tuning-fork in distinguishing, *e.g.* between a tabetic arthro-



pathy and a bone tumour. He agrees that loss or diminution of the sensation argues for a lesion in the posterior columns, instancing the case of Charcot-Marie's amyotrophy (Rydel). He finds the loss, further, often associated with a certain degree of thermo-anæsthesia (*e.g.* in the paraplegias). The probability then is that the paths are very close in the spinal cord, the one in the posterior part of the central grey matter, the other in the anterior part of the posterior columns. In a case of infantile cerebral diplegia, where the lesion was purely cerebral, the author has found serious loss of vibratory sensibility, though sensation otherwise was intact.

He does not commit himself as to the ultimate nature of the sensation.

S. A. KINNIER WILSON.

**DISSOCIATION OF DEEP AND SUPERFICIAL PAIN-SENSATION IN CEREBRAL HEMIPLEGIA.** By LIEPMANN, *Neurol. Centralbl.*, Aug. 16, 1904, p. 740.

THE patient was an idiot girl of 14, suffering from left-sided hemiplegia dating from birth. She was unable to speak or understand. The left upper and lower limbs were spastic, with increased deep reflexes and an extensor plantar response. There was no difference on the two sides in the sensibility to touch or pain. The patient developed an abscess in the dorsum of the left hand. This was incised under local anæsthesia (chloride of ethyl). During the subsequent probing and packing the patient remained placid and showed no signs of pain or discomfort, whilst the lightest pin-pricks in the skin produced pain and made her cry. These phenomena persisted during the fortnight which elapsed before the wound was healed. Liepmann desires to call the attention of surgeons to this observation, in order that they may further investigate the point when operating on the limbs of hemiplegic patients.

PURVES STEWART.

**HYPOTONUS AND HYPERTONUS IN THE SAME PATIENT.** (396) Z. BYCHOWSKI, *Neurol. Centralbl.*, Sept. 1904, p. 786.

BYCHOWSKI gives notes of a case in which the arms showed the typical muscular rigidity (hypertonus) of paralysis agitans, while the legs were markedly hypotonic.

The patient, a woman aged 65, began to suffer from lightning pains in the legs 30 years before she came under observation; her legs gradually became weak and ataxic, so that walking had been impossible for 15 years. Her arms were unaffected until a year

before, when they became gradually weaker and stiffer, and then tremors of the hands appeared ; she also complained much of burning in the back, and constantly required a change of position. The facial expression, the muscular rigidity and position of the arms, the tremors of the hands, were typical of paralysis agitans ; the legs showed very marked hypotonus, absence of knee-jerks and Achilles tendon reflexes, delay and some defect of sensation, especially joint-sensation.

Cases similar to this have been recorded showing a combination of the symptoms of paralysis agitans and tabes. Some authors regard such cases as simply a combination of the two different diseases, while others find an intimate relationship between the symptoms of tabes and of paralysis agitans. Salomonsohn believes that we are justified in looking on a case of this kind as a special clinical entity, for which he proposes the name "tremoparalysis tabiformis."

Bychowski does not care to speculate regarding the explanation of his case, but is content to draw special attention to the diametrically opposite condition of the muscular systems of the upper and lower limbs in one and the same case. (It may be remarked that he has proposed elsewhere to include paralysis agitans in the group of myoses or so-called functional muscular diseases.) He considers that one conclusion is justified, viz., that hypotonus need not affect the whole muscular system in tabes, and that one must recognise some power of independent contraction in muscle.

A. W. MACKINTOSH.

(397) **TREMORS.** F. W. MOTT, *Practitioner*, Sept. 1904, p. 293.

In this lecture, tremors are described as "rhythmical involuntary oscillations of the body, or of separate parts, around their position of equilibrium." Mention is made of the various points to be investigated with regard to tremors, and the attempts at classification are referred to, but "no classification is quite satisfactory." The lecture does not aim at an exhaustive consideration of the subject, but interesting notes are given of the characters of tremors, with specimens of handwriting, occurring in these conditions :—

1. In organic diseases of the nervous system, notably insular sclerosis (in which the tremor "is probably due to an interference with the cerebellar path") and general paralysis.

2. In toxic conditions, *e.g.* from chronic alcoholism, mercury, lead. Stress is laid on the fact that, in many such cases, a neuro-pathic history and abuse of alcohol are important predisposing causes.

3. In infectious diseases, *e.g.* typhoid and typhus fevers.

4. In functional conditions (neuropathic tremors of degenerates)—tremor occurring in several members of a family, progressive, the sole objective sign of the neuropathic state.

5. In neuroses, ( $\alpha$ ) exophthalmic goitre, ( $\beta$ ) paralysis agitans, ( $\gamma$ ) neurasthenia, ( $\delta$ ) hysteria. "There is no form of tremor that may not be simulated by hysteria": special attention is drawn to the great difficulty of distinguishing from disseminated sclerosis.

A. W. MACKINTOSH.

**PARALYSIS OF THE PALATE OF NON-DIPHTHERITIC ORIGIN.**

(398) MERKLEN and BROU, *Arch. Gén. de Méd.*, Aug. 9, 1904.

THE clinical history is given in detail of a case of intense pharyngitis with deposition of false membranes, accompanied with palate paralysis, troubles of accommodation, tachycardia, embryocardia, and slight dyspnoeic crises. Routine bacteriological examination on three several occasions failed to reveal Löffler's bacillus, or indeed rod forms in any quantity, whereas there was always luxuriant streptococcus and staphylococcus formation. Apparently the condition was the result of a toxæmia, and may be compared with the forms of neuritis and myelitis with which the streptococcus is not infrequently associated.

S. A. KINNIER WILSON.

**METAMERIC MANIFESTATIONS IN ZONA AND HYSTERIA.**

(399) CHAVIGNY, *Arch. Gén. de Méd.*, Aug. 2, 1904.

Two hysterical patients, with hemianæsthesia, develop a typical zona corresponding in part to the anæsthetic area. The author talks of a certain "metameric vulnerability," whereby to account for the coincidence. The virus determines the disease, the patient "determines" in what form it is to be manifested.

S. A. KINNIER WILSON.

**THREE CASES OF HEMICEDEMA IN HEMIPLEGIA. LOEPER**

(400) and CROUZON, *Nouv. Icon. de la Salpêtrière*, May-June 1904, p. 181.

VARIOUS vasomotor and trophic disturbances are not uncommon in hemiplegia: but an œdema limited to the affected side is not common, nor is its explanation simple. The tendency has been to account for the condition by a special localisation of the lesion, e.g. the head of the caudate nucleus, the anterior segment of the internal capsule, the internal part of the lenticular nucleus (Parhon). On the other hand, there are œdemas which are

primarily due to renal or cardiac insufficiency, where the onset of a hemiplegia determines the localisation.

In this category are the three cases described by the authors, where several weeks after the ictus, an œdema limited—entirely, in two cases, and practically so in the third—to the affected side appeared, and where albuminuria and cardiac insufficiency pre-existed.

Not all œdemas (in hemiplegia and nervous disease generally) are of renal or cardiac origin. A pure vasotrophic œdema of nervous origin may occur; a pseudo-œdema, known as the "succulent hand" (described by Marinesco in syringomyelia, and by Gilbert and Garnier in hemiplegia), is not infrequent; and there are passing œdemas of neuropathic origin, in peripheral neuritis and in facial neuralgia; what is sometimes described as a "dorsal tumour of the wrist" is the result of musculo-spiral paralysis.

But the cases described are not directly of nervous origin, and the authors invoke as cause the alteration of vasomotor and trophic fibres in hemiplegia which may regulate the distribution of an œdema of visceral origin. The hemiplegia only renders evident an already existing defect of filtration and exchange in interstitial spaces.

S. A. KINNIE WILSON.

**CONTRIBUTION TO THE STUDY OF TROPHŒDEMA.** SAINTON (401) and VOISIN, *Nouv. Icon. de la Salpêtrière*, May-June 1904, p. 189.

In 1901 Meige described, under the name of trophœdema, a white hard painless œdema of one or more segments of either or both lower extremities, persisting throughout life without any particular bearing on the general health. Since then, various cases have been placed on record. The authors report another, concerning a boy of fifteen with the characteristic condition in his right leg, of somewhat rapid development with no absolutely certain cause, though it apparently began not unlike a lymphangitis. The blood in the two limbs was examined: polymorphs, 40-45 %; mononuclears, 43-45 %; lymphocytes, 8-13 %; eosinophils, 2 %.

The remarks on the pathogeny of the disease are of the briefest.

S. A. KINNIE WILSON.

## PSYCHIATRY.

**INSANITY AND EPILEPSY IN RELATION TO LIFE ASSUR-**  
(402) ANOE. Sir WILLIAM R. GOWERS, *Lancet*, Oct. 15, 1904, p. 1061.

GENERAL paralysis causes far more loss to insurance offices than all the other forms of insanity taken together. Indeed, it is sur-

prising to what dimensions the mortality of the assured from insanity is reduced when general paralysis is excluded. Of the fifty-two cases collected by Muirhead, only one exceeded his expectation. The average age at death was forty-four years. Sir William Gowers thinks that the only possible means that could be adopted to lessen this loss are the following:—(1) To ascertain systematically whether or not there has been preceding specific disease; and (2) to ascertain the presence or absence (*a*) of the knee-jerks, and (*b*) of the pupil-light reaction.

No sufferer from acute mania or melancholia could be regarded as insurable, but there are some insane persons who may be taken, of course with a considerable extra, without more risk than insurance offices run every day. Chronic imbecility, if not attended with deterioration of the vital organs, is certainly one of these, provided the patient's means are such as to enable him to secure adequate care. Congenital idiots are not such good lives, as they often develop defects of vitality as life goes on.

There are some cases of paranoia in which a train of delusions has become fixed, has persisted in its limitation, and is innocent in tendency, who may also be regarded as insurable. It is only cases of settled, stationary insanity which can be thought of as possibly eligible.

Cases of incipient, undetermined derangement involve too many risks, and Sir William Gowers is of the opinion that a patient who has recovered from a previous attack of definite insanity is not insurable.

There is a tendency to regard a family history of insanity as almost on a level with that of phthisis in its influence on the eligibility of a life; but the author concludes that in a perfectly healthy proposer no family history of insanity is ground for regarding the life as below the average, so far as death from this cause is concerned.

Suicide constitutes a serious cause of loss to assurance offices. Insurance suicides form a class apart. Insane heredity seems to take very little part in their production, and they are apparently due to financial causes, and imply a strength of will sufficient to enable this mode of exit to be adopted as an escape from the pressure of impending disaster, or as a mode of insuring a secure provision for a family. In this connection it is noteworthy that the suicide of an assured female is almost unknown.

Epilepsy is a rare cause of insurance claims. It is rarely the direct cause of death, even to those who suffer from it, but sometimes indirectly leads to death through accidents which it induces. As regards heredity, the chances are about 1 in 6 that some child of an epileptic parent will present the disease, and that, if he has

reached the age of twenty years without doing so, the chances are perhaps 1 in 50.

Is a proposer who actually suffers from epilepsy insurable? Sir William Gowers is of the opinion that some cases can be accepted, of course at a considerable extra, if they have nearly reached middle life and the character of the disease has shown no tendency to become more grave.

H. DE M. ALEXANDER.

**CHARACTERISTICS OF THE SCOTCH LUNACY SYSTEM.** OWEN  
(403) *Copp, Am. Journ. Insan.*, July 1904, p. 55.

THIS is a short sketchy article but fairly comprehensive, and accurate so far as it goes. The author first touches on the powers of the General Board of Lunacy and its relationship to the District Boards or local authorities, who are the executives for the carrying out of the provisions of the Lunacy Acts in Scotland in its twenty-seven lunacy districts. The supervision of the General Board in matters relating to the admission, detention, and discharge of patients is next dealt with, and the thorough system of registration referred to. "The unique feature of the Scotch regime pertains to family care, in which are 17 per cent. of all registered insane, 20 per cent. of pauper insane"; enough patients to fill six district asylums of average size. The extent of the family care system is due to the active co-operation of the General Board, medical superintendents of asylums, and clerks to the Parish Councils. The saving to parishes in 1902 is reckoned at 30 to 40 per cent. The author describes a visit to several villages in Fife in which this "community care" of the insane is carried out. The author also visited various Royal and District Asylums, and the chief impressions noted are the development of the ideas of "the colony" for custodial care, and of the hospital for curative treatment of recent cases and for research and teaching.

C. C. EASTERBROOK.

**SUGGESTIONS AND PLANS FOR PSYCHOPATHIC WARDS,  
(404) PAVILIONS, AND HOSPITALS FOR AMERICAN CITIES.**

L. PIERCE CLARK, and H. P. ALAN MONTGOMERY, Architect,  
*Am. Journ. Insan.*, July 1904, p. 1.

THE authors give an account of the provision made in Germany for the ordinary hospital treatment of all forms of brain disease accompanied by psychic defect or alienation, such as early and borderland cases of insanity, and the psychoses of epilepsy, hysteria, alcoholism, and the like. In Germany there are three

types of accommodation for such cases: (1) Special wards in general hospitals; (2) special pavilions adjacent to general hospitals; and (3) special hospitals independent of general hospitals, constructed either on the block plan, as at Heidelberg, where the first psychopathic hospital in Germany was built in 1878, and where Kraepelin holds his clinique; or on the separate pavilion or villa plan, as at Kiel, where the latest hospital was opened in 1901, and where Semmerling is director of the clinique. The patients are received into the above places before the certifiable stage, when timeous prophylactic treatment may avert an actual attack of insanity, or for observation previous to certification. At Giessen, in 1896, 6.5 per cent. of the total admissions were not certifiably insane; in 1901, 23 per cent.

The authors then apply their German experience to the case of America, and recommend as follows: (1) Psychopathic wards (one for each sex) in or attached to the general hospital, suitable for cities of 10,000 to 20,000. (2) A psychopathic pavilion, adjacent to but separate from the general hospital, in cities of 20,000 to 50,000. (3) A psychopathic hospital, quite separate and independent from the general hospitals, in larger cities. It should not have more than 150 to 200 beds, including staff. Fourteen plates of plans are given, suggesting the interior and exterior arrangements, for the above three types of accommodation, with estimated cost. Provision is especially made for hydrotherapy and electrotherapy, and for research and teaching.

C. C. EASTERBROOK.

**A CASE OF CATATONIC DEMENTIA PRÆCOX, WITH PSEUDO-  
(405) OEDEMA AND PURPURA.** TREPSAT, *Nouv. Icon. de la Sal-  
pêtrière*, May-June 1904.

PSEUDOEDEMA, according to the researches of Dide, is far from uncommon in dementia præcox of the catatonic type. Trepsat describes a typical case, complicated with purpura. The entire article contains nothing beyond the examination of the patient.

S. A. KINNIER WILSON.

**RADIO-ACTIVE BODIES AND INSANITY.** ROBERT JONES, *Lancet*,  
(406) Oct. 15, 1904.

THE author records the case of a general paralytic aged 28 years, who, after wearing a cap—into the closely quilted seams of which about 500 cubic centimetres of thorium hydroxide were placed—night and day for a period of about three months, was able to leave the asylum "recovered."

H. DE M. ALEXANDER.

**TREATMENT.****NOTE ON THE SERUM TREATMENT OF EXOPHTHALMIC (407) GOITRE.** GEORGE R. MURRAY, *Lancet*, Aug. 27, 1904, p. 583.

OUR present means of treating exophthalmic goitre, and especially those cases in which acute symptoms develop, are unsatisfactory. The evidence we possess tends to show that the symptoms of the disease are due to the toxic effect produced by an excess of thyroidal secretion in the blood, and to counteract this, various endeavours have been made. Portis prepared a *cytotoxic* serum by injecting an emulsion of dog's thyroid into the peritoneal cavity of the goat. Later, when the goat's serum was injected into a dog, it induced degenerative changes in the dog's thyroid gland. Unfortunately degenerative changes were found also in the liver, spleen and kidneys, so that this serum does not seem to be suitable for the treatment of exophthalmic goitre. Möbius employed the serum of a sheep after thyroidectomy with good results, and Lanz has employed the milk of goats after removal of the thyroid gland. The writer endeavoured to prepare an antitoxic serum which would neutralise the effect of the excess of thyroidal secretion. He fed rabbits for some weeks on as large doses of thyroid extract as they could take without undue emaciation. The animals were then killed by bleeding and the serum was used by the mouth in the treatment of two cases of exophthalmic goitre. Improvement took place in certain symptoms in these cases, but it was not more than is obtained by other methods and no specific action could be traced to the serum.

AUTHOR'S ABSTRACT.

**THE BLOOD-SERUM TREATMENT OF EPILEPSY.** H. GERHARTZ, (408) Jr., *Neurol. Centralbl.*, Sept. 16, 1904, p. 835.

GERHARTZ has employed Ceni's injections in two epileptic patients who were undergoing sanatorium treatment. Both cases had previously been under ordinary medicinal treatment in the sanatorium for years. Each patient received ten injections of blood serum in all, during a period of five months—either his own serum previously withdrawn, or that of his fellow-epileptic. Within an hour or two after each injection there was observed a slight increase of polynuclear and transitional leucocytes in the blood, at the expense of the mononuclear and eosinophile cells.

No decided or permanent beneficial effect was produced, as regards the number or severity of the fits, whether during the period of serum injections or afterwards. Occasionally some temporary benefit appeared to result, especially if the injected



serum had been taken from the patient as soon as possible after a severe fit.  
PURVES STEWART.

**THE TREATMENT OF CHRONIC INTERNAL HYDROCEPHALUS (409) BY AUTO-DRAINAGE.** ALFRED G. TAYLOR, N.Y., *Amer. Journ. Med. Sc.*, August 1904.

THE author, after giving a brief account of the pathology of the condition, and the various operative methods that have been attempted to relieve it, describes an operation which he has practised with relatively good results in six cases. The object he has in view is to establish a more or less permanent fistula between the ventricles and the subdural space.

By this method, also, while effectually relieving pressure, he avoids the too rapid reduction of tension, and the great risk of sepsis always present when external drainage is attempted.

The method the author adopts is, after trephining, to pass several strands of forty-day catgut from the subdural space into the ventricle on the right side.

Six cases have been operated on with a mortality of 50 per cent.

Two of the cases have improved distinctly, the mental improvement being most marked, and after a year have not shown any signs of recurrence of increased intra-cranial tension.

In a few brief notes on the cases from a neurological point of view, by Dr Pierce Clark, he lays great stress on the necessity for as early operation as possible, in view of the changes in the nerve cells and developing pyramidal tract from pressure.

DUNCAN LORIMER.

---

## Review

**THE NERVOUS AFFECTIONS OF THE HEART.** Being the Morison Lectures delivered before the Royal College of Physicians in Edinburgh in 1902 and 1903. By GEORGE ALEXANDER GIBSON, M.D., D.Sc., etc. Pp. 99. Edinburgh and London: Young J. Pentland. 1904.

ALL acquainted with Dr Gibson's writings will be prepared to find in these lectures a scholarly knowledge of the literature of the subject which too rarely finds a place in medical dissertations, together with a personal experience of the conditions under discussion which enables the author to weigh in an instructive and judicious manner the results of his own experience and that of

others, and a lucidity of style and wealth of illustration which render the perusal of his writings a pleasure rather than a labour. Dr Gibson has handled a difficult, and, in many respects, obscure subject in a masterly manner, and we know of no monograph on the subject of like compass which will better reward perusal. The author himself, however, would be the first to admit that, while the anatomist has done his part to elucidate the subject (although his task is not by any means finished), and the physiologist has also emulated the assiduity of the anatomist, while the physician—the clinical physiologist—has accumulated much material of practical importance, the ignorance of the essential pathology of the nervous system in heart disease is still abysmal. Many a pathological Curtius must still leap into the abyss before the chasm can be closed, even to the extent to which the anatomical and physiological cracks have been smothered by the work done in these departments. This in itself is not, perhaps, a matter for too severe reprobation, for man is essentially a practical creature, and has not too much time to work for that which he imagines profiteth not, or in fields in which the current notions of physiologists seem to indicate that the quest is likely to be tedious and the results incommensurate with the labour expended. By the routine pathologist of the dead-house, the cardiac nervous system, it will not be denied, is practically, although not altogether ignored. Consequently, there is of necessity room for much speculation in any consideration of the nervous affections of the heart. Notwithstanding this acknowledged disadvantage, Dr Gibson has compressed into these six lectures much information of immediate practical value, and has also suggested food for present thought and future work. He has divided his subject into the two great classes of sensory and motor disturbances, dealing with the former in its clinical, pathological and therapeutic aspects; and with the latter in regard to the cardinal conditions of rate, rhythm and force. Within the compass suitable in such a review as this, it would manifestly be impossible to refer specially to much of the important material laid before the reader, and after a careful perusal of the work we can only touch upon certain points. Dr Gibson expresses a strong and rational objection to the employment of the terms "true" and "false" angina pectoris, preferring to these the terms "organic" and "inorganic" or "functional." This is possibly the better expressed meaning of those who have used the terms objected to, for, false or spurious angina pectoris can only correctly denote pain in the chest unconnected with the heart. The author most usefully dwells upon the serious nature of some cases in which discomfort and a sense of oppression never rise to the height of the classical agony of Heberden's disease. Among the causes provocative of the inorganic variety of angina pectoris and of disordered cardiac rhythm, he

emphasises the pernicious influence of tobacco. That the tobacco heart is a clinical entity, the existence of which cannot be denied, is indisputable, but when one considers the vast amount of tobacco injudiciously consumed in the country, and the comparative rarity especially of sensory disorders directly attributable to it, we are not quite certain that a calnative influence and solace is not frequently withdrawn from patients without effecting good proportionate with the self-denial imposed when tobacco is altogether interdicted. The genial author, who disclaims much personal acquaintance with the pernicious weed (p. 77), appears indeed to us to write as though he contemplated issuing a modern version of the "Counterblast"! We trust we do not misrepresent him.

The pathological section is of necessity largely anatomical, physiological and semeiological, and the author recognises an unavoidable overlapping of these considerations in treating his subject in the present state of our knowledge. With Mackenzie and some others he regards Nothnagel's vaso-motor variety of angina as of rare occurrence, but points out the more common incidence of arterial spasm in cases of cardiac failure from various causes. The surface representation of visceral disorders is somewhat fully treated. The section dealing with the *treatment* of sensory disorders will well repay perusal, and is evidently based upon an intimate knowledge of the views of others and a large personal experience.

The difficult question of the nature and treatment of motor disturbances is dealt with in a masterly manner, while the graphic illustrations which the author exhibits in his text to elucidate these obscure states add practical value to the lectures as a medium for the instruction of those less fully conversant with the subject. The remarks upon the paradoxical pulse are no doubt in accordance with prevalent views upon the subject; but we confess to entertaining a heresy upon this point, based, as it has appeared to us, upon unmistakable experience. "Under ordinary circumstances it is found," writes Dr Gibson, "that with inspiration the pulse becomes stronger and more frequent." We agree that it becomes more frequent, but not, we believe, stronger. Indeed, increased rate is usually associated in our experience with diminished force, that is, with a smaller arterial wave. This fact appears to us to be borne out by the sphygmograms given, and, as we have observed the same fact under normal circumstances—which the author states he has *not* met with—the term in question has always appeared to us a misnomer. It would be paradoxical, in our view, if the expiratory phase of respiration were not also associated with fuller and stronger arterial pulsation.

The final and important section on cardiac "force," in which

Dr Gibson, with his usual knowledge and courtesy, does full justice to the work of M'William, should be carefully read, while his remarks upon the treatment of the serious contingency of syncope have practical value. In this connection, however, we should have been glad had he emphasised more strongly the value of artificial respiration. Dealing, however, as was his task, and within a limited time, with so large a subject, he has doubtless had to compress his material more than he would have done had more time been at his disposal. We have only to repeat our conviction that these lectures provide, within their well and clearly printed pages, a more instructive account of the conditions with which they deal than any other work of similar size known to us, and that they are calculated to enhance the well-established reputation of their author for comprehensive thought and lucid expression.

## Bibliography

### ANATOMY

- GURWITSCH. Morphologie und Biologie der Zelle. G. Fischer, Jena 1904, M. 10.  
 FRANZ NISSEL. Histologische und histopathologische Arbeiten über die Grosshirnrinde. Gustav Fischer, Jena, 1904, 42a.  
 F. GRIFFITH and W. B. WARRINGTON. On the Cells of the Spinal Ganglia. *Brit. Med. Journ.*, Sept. 24, 1904, p. 752.  
 G. PANEGROSSI. Weiterer Beitrag zum Studium der Augenmuskelnervenkerne. *Monatsschr. f. Psychiat. u. Neurol.*, Bd. xvi., H. 3, 1904, S. 344.  
 AZOULAY. Les neurofibrilles d'après la méthode et les travaux de S. Ramon y Cajal. *Presse méd.*, oct. 5, 1904, p. 835.  
 GUISEPPE LEVI. Ueber die Entwicklung und Histogenese der Ammonshornformation. *Arch. f. Mikros. Anat.*, Bd. lxi., H. 3, 1904, S. 389.  
 R. BING. Beitrag zur Kenntniss der endogenen Rückenmarksfasern beim Menschen. *Arch. f. Psychiat.*, Bd. xxxix., H. 1, 1904, S. 74.  
 HÖSEL. Ueber die Markkreisung der sogenannten Körperfühlsphäre und der Reich- und Sehstrahlung des Menschen. *Arch. f. Psychiat.*, Bd. xxxix., H. 1, 1904, S. 195.  
 LAIGNEL-LAVASTINE. Les variations macroscopiques du plexus solaire. *Bull. et mémoires de la Soc. Anat. de Paris*, Bd. lxxxix., T. vi., 1904, S. 885.  
**COMPARATIVE ANATOMY**—  
 E. CORDS. Beiträge zur Lehre von Kopfnervensystem der Vögel. *Anat. Hefte*, Abteil. i., H. 78, 1904, S. 49.  
 EDGAR A. BEDFORD. The Early History of the Olfactory Nerve in Swine. *Journ. Comp. Neurol. and Psychol.*, Sept. 1904, p. 390.  
 VICTOR E. EMMEL. The Relation of the Chorda Tympani to the Visceral Arches in *Microtus*. *Journ. Comp. Neurol. and Psychol.*, Sept. 1904, p. 411.

### PHYSIOLOGY

- J. E. DONLEY. On the Early History of Cerebral Localization. *Am. Journ. Med. Sc.*, Oct. 1904, p. 711.  
 A. G. GIBSON. Recent Work on the Functions of the Spinal Cord. *Clin. Journ.*, Sept. 28 and Oct. 5, 1904, pp. 375, 396.  
 PIERRE BONNIER. Schéma bulbaire. *Rev. Neurol.*, oct. 15, 1904, p. 993.  
 U. SCHEVEN. Ueber den Einfluss der Anämie auf die Erregbarkeit der weissen Substanz des Centralnervensystems. *Arch. f. Psychiat.*, Bd. xxxix., H. 1, 1904, S. 189.

- KÖSTER. Experimenteller Beitrag zur Physiologie der Spinalganglien und zur Lehre von den trophischen Nerven. Engelmann, Leipzig, 1904, M. 6.  
 G. ABELSDORFF und A. FEILCHENFELD. Erwiderung auf die vorstehenden Bemerkungen von Dr H. Wolff. *Ztschr. f. Psychol. u. Physiol. d. Sinnesorgane*, Bd. xxxvi., H. 1 u. 2, 1904, S. 98.  
 HUGO WOLFF. Bemerkungen zu der Arbeit "Über die Abhängigkeit der Pupillarreaktion von Ort und Ausdehnung der dereinsten Netzhautfläche." *Ztschr. f. Psychol. u. Physiol. d. Sinnesorgane*, Bd. xxxvi., H. 1 u. 2, 1904, S. 93.  
 CHAUVEAU. Le travail musculaire et sa dépense énergétique dans la contraction dynamique. *Acad. des sciences*, juillet 26, 1904, p. 1912.  
 F. P. SGOBBO. Manifestations électriques dues au travail musculaire. *Ann. d'Electrobiol.*, Vol. vii., No. 4, 1904, p. 404.  
 GUILLOZ. Sur la détermination quantitative de l'excitabilité électrique de muscles altérés restés longtemps inactifs. *Soc. de biol.*, juillet 22, 1904, p. 153.  
 G. GALEOTTI. Modifications du réflexe de la déglutition étudiée dans la Capanna Regina Margherita. *Arch. Ital. de Biol.*, Vol. xli., f. iii., 1904, p. 875.

## PSYCHOLOGY

- DE SANCTIS. Il problema della coscienza nella Psicologia scientifica. *Ann. d. Instit. Psichiat. d. r. Univ. di Roma*, Vol. iii., f. 1, 1904, p. 35.  
 GODFERNAX. Le parallélisme psychophysique et ses conséquences. *Rev. Philosophique*, oct. 1904, p. 329.  
 HENRY RUTGERS MARSHALL. "Automatic" and Intelligent Activities. *Journ. Nerv. and Ment. Dis.*, Sept. 1904, p. 588.  
 A. E. TAYLOR. Mind and Body in Recent Psychology. *Mind*, Oct. 1904, p. 476.  
 HERRICK. Recent Contributions to the Body-Mind Controversy. *Journ. Comp. Neurol. and Psychol.*, Sept. 1904, p. 421.  
 HANS BERGER. Über die körperlichen Äusserungen psychischer Zustände. Weitere experimentelle Beiträge zur Lehre von der Blutzirkulation in der Schädelhöhle des Menschen. G. Fischer, Jena, 1904, M. 20.  
 W. JAMES. Humanism and Truth. *Mind*, Oct. 1904, p. 457.  
 H. E. JOST. (1) Ueber die beste Art, geistig zu arbeiten; (2) Ueber Gedächtnisbildung. *Arch. f. d. gesam. Psychol.*, Bd. iii., H. 4, 1904, S. 243.  
 STILLING. Psychologie der Gesichtsvorstellung nach Kants Theorie der Erfahrung. *Arch. f. d. gesam. Psychol.*, Bd. iii., H. 4, 1904, S. 243.  
 HOFER und ST WITASEK. Hundert Psychologische Schulversuche mit Angabe der Apparate. *Arch. f. d. gesam. Psychol.*, Bd. iii., H. 4, 1904, S. 242.  
 MAX LEVY. Ueber die Helligkeitsverteilung im Spektrum für das helladaptierte Auge. *Ztschr. f. Psychol. u. Physiol. d. Sinnesorgane*, Bd. xxxvi., H. 1 u. 2, 1904, S. 74.  
 F. KIESOW. Zur Frage nach den Schmeckflächen des hinteren kindlichen Mundraumes. *Ztschr. f. Psychol. u. Physiol. d. Sinnesorgane*, Bd. xxxvi., H. 1 u. 2, 1904, S. 90.  
 ERICH BECHER. Experimentelle und kritische Beiträge zur Psychologie des Lesens bei kurzen Expositionszeiten. *Ztschr. f. Psychol. u. Physiol. d. Sinnesorgane*, Bd. xxxvi., H. 1 u. 2, 1904, S. 19.  
 LÖESER. Ueber den Einfluss der Dunkel-adaptation auf die spezifische Farbenschwelle. *Ztschr. f. Psychol. u. Physiol. d. Sinnesorgane*, Bd. xxxvi., H. 1 u. 2, 1904, S. 1.  
 K. MARBE. Ueber den Rhythmus der Prosa. *Arch. f. d. gesam. Psychol.*, Bd. iii., H. 4, 1904, S. 244.  
 ERDMANN. Drei Beiträge zu einer allgemeinen Theorie der "Begriffe." *Arch. f. d. gesam. Psychol.*, Bd. iii., H. 4, 1904, S. 244.  
 H. DIELS. Die Fragmente der Vorsokratiker. *Arch. f. d. gesam. Psychol.*, Bd. iii., H. 4, 1904, S. 245.  
 R. ÖHLER. Friedrich Nietzsche und die Vorsokratiker. *Arch. f. d. gesam. Psychol.*, Bd. iii., H. 4, 1904, S. 247.  
 R. RICHTER. Friedrich Nietzsche. Sein Leben und sein Werk. *Arch. f. d. gesam. Psychol.*, Bd. iii., H. 4, 1904, S. 252.  
 O. FLÜGEL. Das Ich und die sittlichen Ideen im Leben der Völker. *Arch. f. d. gesam. Psychol.*, Bd. iii., H. 4, 1904, S. 254.  
 W. M. BOWACK. Another View of Industrialism. *Arch. f. d. gesam. Psychol.*, Bd. iii., H. 4, 1904, S. 255.  
 T. K. ABBOTT. Fresh Light on Molyneux' Problem: Dr Ramsay's Case. *Mind*, Oct. 1904, p. 543.  
 B. RUSSELL. Meinong's Theory of Complexes and Assumptions (III.). *Mind*, Oct. 1904, p. 509.  
 F. S. C. SCHÜLLER. In Defence of Humanism. *Mind*, Oct. 1904, p. 525.

## PATHOLOGY

- R. KENNEDY. On the Histological Changes occurring in Ununited Divided Nerve. *Brit. Med. Journ.*, Sept. 24, 1904, p. 729.
- S. WALTER RANSON. Retrograde Regeneration in the Corpus Callosum of the White Rat. *Journ. Comp. Neurol. and Psychol.*, Sept. 1904, p. 381.
- GEORGE LAMB and WALTER K. HUNTER. On the Action of Venoms of Different Species of Poisonous Snakes on the Nervous System. *Lancet*, Oct. 22, 1904, p. 1146.
- HUBERT M. TURNBULL. Bilateral Loss of Postcentral Cortex, apparently Congenital, in an Adult. *Brain*, Summer 1904, p. 209.
- A. LÉRI. Étude sur le nerf optique dans l'amaurose tabétique. *Nouv. Icon. de la Salpêtrière*, sept.-oct. 1904, p. 358.
- CARL HUDOVERNIG. Mikroskopische Veränderungen im Vaguskerne in einem Falle von Oesophaguscarcinom. *Neurol. Centralbl.*, Oct. 16, 1904, S. 935.
- SIEBENMANN. Pathologische Anatomie und Pathogenese der Taubstummheit. Bergmann, Wiesbaden, 1904, M. 3.
- HÖLDER. Pathologische Anatomie der Gehirnerschütterung beim Menschen. Weise, Stuttgart, 1904, M. 5.
- DAVID ORR and R. G. ROWS. Lesions of the Posterior Columns in General Paralysis. *Brit. Med. Journ.*, Oct. 15, 1904, p. 978.
- THOMAS et HAUSER. Histologie pathologique et pathogénie de la Syringomyélie. *Nouv. Icon. de la Salpêtrière*, sept.-oct. 1904, p. 376.
- CATOLA. Note sur les concrétions calcaires dans le cerveau. *Nouv. Icon. de la Salpêtrière*, sept.-oct. 1904, p. 354.
- ZINGERLE. Erwiderung zu dem Aufsatz von Dr O. von Leonowa—v. Lange: Zur pathologischen Entwicklung des Centralnervensystems. *Arch. f. Psychiat.*, Bd. xxxix., H. 1, 1904, S. 430.

## CLINICAL NEUROLOGY AND PSYCHIATRY

## GENERAL—

- F. SAVARY PEARCE. A Practical Treatise on Nervous Diseases. Sydney Appleton, London, 1904, 14s.
- BABINSKI. Introduction à la sémiologie des maladies du système nerveux. Des symptômes objectives que la volonté est incapable de reproduire. De leur importance en médecine légale. *Gaz. de Hôp.*, oct. 11, 1904, p. 1125.
- ARIA L. DERDIGER. Nervous Diseases and Eye-Strain. *Chicago Med. Recorder*, Aug. 15, 1904.
- HUGO RAUBITSCHKE. Zur Tuberkulose des Zentralnervensystems. *Wien. Klin. Wchnschr.*, Sept. 29, 1904, S. 1033.

## MUSCLES—

- CESTAN et LEJONNE. Un cas de myopathie avec rétractions: examen anatomique. *Nouv. Icon. de la Salpêtrière*, sept.-oct. 1904, p. 343.

## PERIPHERAL NERVES—

- LORTAT-JACOB et SABARÉANU. Sciatique radiculaire unilatérale. *Presse méd.*, oct. 5, 1904, p. 633.
- THIELLE. Algies sciatiques traitées par le courant ondulatoire. *Ann. d'Électro-biol. et de Radiol.*, No. 3, 1904, p. 323.
- P. SAINTON et J. CASTAIGNE. Coexistence de la cirrhose alcoolique et de névrites périphériques. *Arch. gén. de méd.*, sept. 27, 1904, p. 2447.
- LEMASSON. Névrites périphériques au cours des cirrhoses alcooliques. *Thèse de Paris*, 1904.
- BERTHELOT. De la gravité des paralysies diphtériques précoces. *Thèse de Paris*, 1904.
- COMBY. Paralysies diphtériques guéries par le sérum. *Arch. méd. des enfants*, juillet 1904, p. 411.
- P. N. GERRARD. Beri-Beri: its Symptoms and Symptomatic Treatment. J. & A. Churchill, London, 1904, 2s. 6d.
- Herpes Zoster.**—VORNER. Ueber wiederauftretenden Herpes Zoster, im besondern über Zoster erythematosus und Zoster vegetans. *München. med. Wchnschr.*, Sept. 27, 1904, S. 1734.

## SPINAL CORD—

- Tabes.**—H. DOUGLAS SINGER. The Diagnosis and Treatment of Early Tabes. *Western Med. Rev.*, Lincoln, Aug. 1904.
- LOEB. Ein Fall von Atemstillstand bei Tabes. *München. med. Wchnschr.*, Oct. 11, 1904, S. 1823.
- REICHARDT. Das Verhalten des Rückenmarkes bei reflectorischer Pupillenstarre. *Arch. f. Psychiat.*, Bd. xxxix., H. 1, 1904 S. 324.

- Disseminated Sclerosis.**—EDUARD MÜLLER. Die multiple Sklerose des Gehirns und Rückenmarks. G. Fischer, Jena, 1904, 10s. 6d.
- ERNEST S. REYNOLDS.** Some Cases of Family Disseminated Sclerosis. *Brain*, Summer 1904, p. 163.
- Syringomyelia.**—S. A. KINNIER WILSON. Un cas de syringomyélie und syringobulbia. Dégénération du ruban de Reil. *Rev. de méd.*, sept. 10, 1904, p. 686.
- Tumour.**—G. WOOLSEY. A Case of Tumour of the Spinal Cord. *Med. News*, Oct. 1, 1904, p. 625.
- Abscess.**—W. ALDREN TURNER and JAMES COLLIER. Intramedullary Abscess of the Spinal Cord. An Account of Three Cases. *Brain*, Summer 1904, 199.
- Injury.**—A. H. TUBBY. A Clinical Lecture on Injuries to the Spinal Column and Spinal Cord. *Clin. Journ.*, Oct. 5, 1904, p. 385.
- ERNEST RINGROSE.** A Case of Fracture-Dislocation of the Cervical Vertebrae. *Lancet*, Oct. 15, 1904, p. 1076.
- Brown-Sequard Symptom Complex.**—SFORZA. Sulla sindrome di Brown-Sequard da lesione traumatica (Contributo clinico). *Ann. d. Instit. Psichiat. d. r. Univ. di Roma*, Vol. iii., f. 1, 1904, p. 119.
- SCIAMANNA.** Sindromi di Brown-Sequard d'origine riflessa. *Ann. dell' Instit. psichiat. d. r. Univ. di Roma*, Vol. iii., f. 1, 1904, p. 3.
- Spine.**—G. C. STEELE-PERKINS. A Note on the Pathology of Lateral Curvature of the Spine. *Lancet*, Sept. 24, 1904, p. 894.

#### BRAIN AND MENBRANES—

- Meningitis.**—S. S. KNEASS and JOSEPH SAILER. A Case of Tuberculous Meningitis, with Secondary Infection. *Journ. Nerv. and Ment. Dis.*, Oct. 1904, p. 660.
- Hydrocephalus.**—JAULIN. Un cas d'Hydrocéphalie aiguë. *Ann. méd.-chir. du Centre*, juillet 10, 1904, p. 347.
- Hæmorrhage.**—WILLIAM C. KRAUSS. Traumatic Hæmorrhage over the Third Anterior Frontal Convolution. *Am. Journ. Med. Sc.*, Sept. 1904, p. 393.
- E. E. SOUTHARD and F. R. SIMS.** A Case of Cortical Hæmorrhages following Scarlet Fever. *Journ. Amer. Med. Assoc.*, Sept. 17, 1904, p. 789.
- PIERCE BAILEY.** Traumatic Apoplexy. *Med. Record*, Oct. 1, 1904, p. 528.
- Hemiplegia.**—BOUCHAUD. Hémiplegie cérébrale infantile et hémistaxie. *Rev. Neurol.*, oct. 15, 1904, p. 996.
- Tumour.**—F. H. HOWARD and E. E. SOUTHARD. A Case of Glioma in the Sella Turcica. *Am. Journ. Med. Sc.*, Oct. 1904, p. 679.
- F. FRANCESCHI.** Il sonno patologico nei tumori cerebrali. *Riv. di Patolog. nerv. e ment.*, Vol. ix., f. 10, 1904, p. 457.
- FRASER.** A Case of Complete Transposition of the Viscera, with Cerebral Tumour and other Pathological Conditions. *Edinburgh Med. Journ.*, Oct. 1904, p. 295.
- Syphilis.**—KUF'S. Beitrag zur Syphilis des Gehirns und der Hypophysis und zur Differential-diagnose zwischen der Tuberculose und Syphilis des Centralnervensystems. *Arch. f. Psychiat.*, Bd. xxxix., H. 1, 1904, S. 184.
- Abscess.**—PERCY S. WEBSTER. Two Intra-cranial Abscesses, with Sinus Phlebitis. *Intercolonial Med. Journ. of Australia*, Vol. ix., No. 8, 1904, p. 409.
- Sinus Thrombosis.**—BARTON H. POTTS. Report of an Anomalous Lateral Sinus. Report of a Case of Thrombosis of the Lateral Sinus. *Am. Journ. Med. Sc.*, Oct. 1904, p. 676.
- W. G. SPILLER and C. D. CAMP.** Autochthonous Sinus Thrombosis of the Cerebral Dura. *Journ. Amer. Med. Assoc.*, Sept. 24, 1904, p. 885.
- General Paralysis.**—REUTSCH. Ueber zwei Fälle von Dementia paralytica mit Hirsnsyphilis. *Arch. f. Psychiat.*, Bd. xxxix., H. 1, 1904, S. 181.
- CH. CORNILLLOT.** De la perte de mémoire du calcul comme signe précoce de l'affaiblissement intellectuel chez les paralytiques généraux. *Thèse de Paris*, Jouve, 1904.
- G. PERUSINI.** Sopra un caso di paralisi progressiva infanto juvenilis simulante la sindrome data da una atrofia cerebello spinale (Contributo anatomo-patologico e clinico). *Ann. d. Institut. Psichiat. d. r. Univ. di Roma*, Vol. iii., f. 1, 1904, p. 153.
- O. FISCHER.** Zur Frage der Zytodiagnose der progressiven Paralyse. *Prag. med. Wchnschr.*, Oct. 6, 1904, S. 515.
- Cerebral Diplegia.**—MEIZONNET. De la maladie de Little et de son traitement, surtout orthopédique. *Sidobre et Gabarel*, Cette, 1904.

#### GENERAL AND FUNCTIONAL DISEASES—

- Chorea.**—MALCOLM MACKAY. Hereditary Chorea in Eighteen Members of a Family. *Med. News*, N.Y., Sept. 10, 1904.

- J. J. S. LUCAS. The Relationship of Chorea and Rheumatism. *British Med. Jour.*, Vol. xii, No. 86, 1904, p. 239.
- MORRIS J. LEWIS and W. T. LONGCOPE. Experimental Arthritis and Endocarditis, produced by a Streptococcus isolated from the Blood of a Case of Rheumatism, Endocarditis, and Chorea. *Am. Jour. Med. Sc.*, Oct. 1904.
- VASCHIDE et VURPAS. Essai sur la physiologie pathologique du mouvement. Disparition des mouvements dans la chorée chronique. *Rev. de Méd.*, sept. 10, 1904, p. 704.
- Epilepsy.**—WILLIAM P. SPRATLING. Epilepsy and its Treatment. W. B. Saunders & Co., London, Phila., and New York, 1904, 18s.
- NINO SAVAJA. Le siège des convulsions épileptiformes toniques et cloniques. *Ann. d'Électrobiol. et de Radiol.*, Nos. 3 and 4, 1904, pp. 257, 455.
- GUIDI e GUERRI. Sul ricambio materiale degli epilettici. *Ann. d. Instit. Psichiat. d. r. Univ. di Roma*, Vol. iii, f. 1, 1904, 67.
- M. L. BIANCHINI. "Aura canora" épileptique et équivalents musicaux. *Rev. Neurol.*, Sept. 30, 1904, p. 970.
- A. ZIMMERN et G. DIMIER. De l'épilepsie expérimentale. *Arch. d'Élect. méd.*, oct. 10, 1904, p. 741.
- GUSTAVE GOUIN. Étude de l'épilepsie expérimentale pas les courants intermittents de basse tension. *Ann. d'Électrobiol.*, Vol. vii, 1904, Nos. 3 and 4, pp. 289, 422.
- Neurasthenia.**—G. A. SUTHERLAND. A Lecture on Albuminuria and Neurasthenia at Puberty. *Clin. Jour.*, Oct. 12, 1904, p. 410.
- Hysteria.**—HELLPACH. Grundlinien einer Psychologie der Hysteria. Engelmann, Leipzig, 1904, M. 10.
- CERLETTI. Sulle recenti concezioni dell'isteria e della suggestione a proposito di una endemia di possessione demoniaca. *Ann. d. Instit. Psichiat. d. r. Univ. di Roma*, Vol. iii, f. 1, 1904, p. 92.
- S. WEIR MITCHELL and W. G. SPILLER. A Case of Uncomplicated Hysteria in the Male, lasting Thirty Years, with Post-mortem Examination. *Journ. Nerv. and Ment. Dis.*, Oct. 1904, p. 625.
- ADOLPH O. PFINGST. Some Phases of Hysteria as we see it in the Study of Diseases of the Eye, Ear, Nose and Throat. *Chicago Med. Recorder*, Aug. 15, 1904.
- WEILL-HALLÉ. La constitution physique dans l'hystérie infantile. *Rev. mens. des mal. de l'enfance*, oct. 1904, p. 445.
- J. K. FRIEDJUNG. Eine typische Form der Hysteria des Kindesalters und ihre Beziehung zu der Anatomie der Linea alba. *Ztschr. f. Heilk.*, Bd. xxv., H. 9, 1904, S. 209.
- COTTU. L'hystéro-traumatisme cardiaque. *Thèse de Paris*, 1904.
- J. VALOBRA. Emianopsia isterica. *Riv. crit. di Clin. Med.*, Sett. 17 e 24, 1904, pp. 605, 621.
- Tetanus.**—POAN de SAPINCOURT. Du tétanos céphalique à paralysie faciale. *Thèse de Paris*, 1904.
- Migraine.**—HENRY MEIGE. Migraine ophthalmique, hémianopsie et aphasie transitoires. Hémiface succulente. Photophobie et tic de clignement. *Rev. Neurol.*, sept. 30, 1904, p. 961.
- Neuralgia.**—A. ZIMMERN. Le traitement électrique de la névralgie faciale; perfectionnement apporté à la méthode. *Arch. d'Élect. méd.*, sept. 26, 1904, p. 698.
- Tic Convulsif.**—P. D. SICCARDI. Considerazioni su la fisiopatologia e su la teoria del tic. Marchetti, Ancona, 1904.
- HENRY MEIGE et E. FEINDEL. Remarques cliniques et thérapeutiques sur quelques tics de l'enfance. *Journ. de Neurol.*, sept. 20, 1904, p. 841.
- Exophthalmic Goitre.**—LAIGNEL-LAVASTINE et P. BLOCH. Syndrome de Basedow chez une tuberculeuse. *Arch. gén. de méd.*, sept. 27, 1904, p. 2456.
- Adiposa Dolorosa.**—A. HONÉE. De la maladie de Dercum (adiposa douloureuse généralisée). *Thèse de Paris*, 1904.
- ALCOHOLISM, POISONS, ETC.**—
- RICHARD C. CABOT. The Relation of Alcohol to Arterio-sclerosis. *Journ. Amer. Med. Assoc.*, Sept. 17, 1904, p. 772.
- T. N. KELYNACK. On the Study of Alcoholism. *Brit. Journ. of Inebriety*, Oct. 1904, p. 64.
- T. D. CROTHERS. Alcoholism and Inebriety: An Etiological Study. *Brit. Journ. of Inebriety*, Oct. 1904, p. 69.
- HARRY CAMPBELL. The Pathology of Chronic Alcoholism: A Criticism. *Brit. Journ. of Inebriety*, Oct. 1904, p. 64.
- VASCHIDE et DRACHICESCO. La psychologie de l'alcoolisme d'après les travaux de l'École de Kraepelin. *Arch. gén. de méd.*, oct. 11, 1904, p. 2681.



- WILLIAM C. SULLIVAN. The Criminal Responsibility of the Alcoholic. *Brit. Journ. of Inebriety*, Oct. 1904, p. 48.
- FRED. C. JOHNSTON. The Treatment of Acute Alcoholism. *Therapeutic Gaz.*, Detroit, Aug. 15, 1904.
- H. W. MITCHELL. Alcoholism and its Treatment among the Insane. *Therapeutic Gaz.*, Detroit, Aug. 15, 1904.
- JOSEPH COLLINS. The Treatment of Alcoholism in the City Hospital of New York. *Therapeutic Gaz.*, Detroit, Aug. 15, 1904.
- G. H. R. DABBS. Either or Neither: A Note on the Treatment of Inebriety. *Brit. Journ. of Inebriety*, Oct. 1904, p. 77.

#### SPECIAL SENSES AND CRANIAL NERVES—

- C. E. BEEVOR and JAMES COLLIER. A Contribution to the Study of the Cortical Localisation of Vision. A Case of Quadrantic Hemianopia with Pathological Examination. *Brain*, Summer 1904, p. 155.
- W. UHTHOFF. Zur Frage der Stauungspapille. *Neurol. Centralbl.*, Oct. 16, 1904, S. 930.
- PISSAREFF. Quelques réflexions sur la stase pupillaire, à propos de deux cas de tumeur cérébrale. *Delord-Boehm et Martial*, Montpellier, 1904.
- FABRIZI. Contributo clinico allo studio delle paralisi oculari. *Ann. d. Instit. Psichiat. d. r. Univ. d. Roma*, Vol. iii., f. 1, 1904, p. 46.
- ERNST BLOCH. Ein Fall von Abducenslähmung und Extremitätenparese nach Schädelbruch. *Neurol. Centralbl.*, Oct. 16, 1904, S. 940.
- BENOIT. Des troubles moteurs oculaires dans les maladies de l'oreille. Schneider, Lyon, 1904.
- DEHERAIN. L'égéralité pupillaire dans les maladies du poulmon et de la plèvre. *Presse méd.*, oct. 1, 1904, p. 630.
- ALESSANDRO MARINA. Sul restringimento pupillare che si osserva ai movimenti laterali dei bulbi, nell'occhio che va all'interno. *Polioclinico*, Vol. xi.-M., 1904.

#### MISCELLANEOUS SYMPTOMS—

- NARDELLI. Il limite tra la sensibilità termica e dolorifica. *Ann. d. Instit. Psichiat. d. r. Univ. di Roma*, Vol. iii., f. 1, 1904, p. 227.
- MARTIN. Die Rückenmarksanästhesie bei Gebärenden. *München. med. Wchnschr.*, Oct. 11, 1904, S. 1817.
- SPILLER and WEISENBURG. A Further Study on the Sensory Segmental Zone of the Umbilicus. *Rev. Neurol. and Psychiat.*, Oct. 1904, p. 681.
- STRÜMPPELL. Ueber die Bedeutung der Sensibilitätsprüfungen mit besonderer Berücksichtigung des Drucksinnes. *Deutsche med. Wchnschr.*, Sept. 22 u. 29, 1904, S. 1411 u. 1460.
- GAUSSEL. Spasme bilatéral des muscles du cou et de la face. *Nouv. Icon. de la Salpêtrière*, sept.-oct., 1904, p. 337.
- F. PARKES WEBER. Fæcal Vomiting and Reversed Peristalsis in Functional Nervous (Cerebral) Disease: A Summary of Cases and Conclusions. *Brain*, Summer 1904, p. 170.
- ISADOR H. CORIAT. Reduplicative Paramnesia. *Journ. Nerv. and Ment. Dis.*, Sept. 1904, p. 577.
- C. K. MILLS. Aphasia and the Cerebral Zone of Speech. *Am. Journ. Med. Sc.*, Sept. 1904, pp. 875, 639.
- HUGO STRAUSS. Ueber angiospastische Gangrän (Raynaud'sche Krankheit). *Arch. f. Psychiat.*, Bd. xxxix., H. 1, 1904, S. 109.

#### MENTAL DISEASES—

- W. R. GOWERS. An Address on Insanity and Epilepsy in Relation to Life Assurance. *Lancet*, Oct. 15, 1904, p. 1061.
- W. KÖNIG. The Problem of Heredity from the Psychiatric Aspect. *Brit. Med. Journ.*, Oct. 15, 1904, p. 965.
- J. BEARD. Heredity in its Biological and Psychiatric Aspects. *Brit. Med. Journ.*, Oct. 15, 1904, p. 963.
- HAHNLE. Der heutige Stand der Erbliehkeitsfrage in der Neuro- und Psychopathologie. *Neurol. Centralbl.*, Oct. 1, 1904, S. 882.
- C. WERNICKE. Outlines of Clinical Psychiatry in Clinical Lectures. *Alienist and Neurologist*, Aug. 1904.
- E. MEYER. Ueber Autointoxications-psychosen. *Arch. f. Psychiat.*, Bd. xxxix., H. 1, 1904, S. 286.
- W. WARD. Zur Geschichte und Kritik der sogenannten psychischen Zwangszustände. *Arch. f. Psychiat.*, Bd. xxxix., H. 1, 1904, S. 239.
- E. G. YOUNGER. Insanity in Everyday Practice. Baillière, Tindall & Cox, London, 1904, 2s. 6d.

- VIGOUROUX et JUQUELIER. La Contagion Mentale. Doin, Paris, 1904, 4 fr.
- ALICOT. Contribution à l'urologie clinique des maladies mentales. Firmin, Montane et Sicard, Montpellier, 1904.
- LOMPET et LEBRET. Délire inanition. *Presse méd.*, oct. 1, 1904, p. 625.
- W. ALTER. Stereopsychosen. *Monatsschr. f. Psychiat. u. Neurol.*, Bd. xvi, H. 3, 1904, S. 297.
- ERNEST C. REYER. Paretic Dementia. *Indiana Med. Journ.*, Aug. 1904.
- W. WEYGANDT. Alte Dementia praecox. *Centralbl. f. Nervenheilk. u. Psychiat.*, Oct. 15, 1904, S. 613.
- CONOLLY NORMAN. Dementia Praecox. *Brit. Med. Journ.*, Oct. 15, 1904, p. 972.
- E. C. BROWN. Paranoia. *Cleveland Med. Journ.*, Sept. 1904.
- KÖLPIN. Klinische Beiträge zur Melancholiafrage. *Arch. f. Psychiat.*, Bd. xxxix, H. 1, 1904, S. 1.
- KNAPP. Spastische Symptome bei funktionellen Geistesstörungen. *Monatsschr. f. Psychiat. u. Neurol.*, Bd. xvi, H. 3, 1904, S. 327.
- F. PROBST. Der Fall Otto Weininger: Eine psychiatrische Studie. Bergmann, Wiesbaden, 1904, M. 2.
- BERTHA DOWNING. Mental Defectives. *Boston Med. and Surg. Journ.*, Sept. 1, 1904.
- SCHOTT. Ueber Simulation von Geistesstörung. *München. med. Wchnschr.*, Oct. 18, 1904, S. 1869.
- L. W. WEBER. Die Lebensdauer der Geisteskranken. *Ztschr. f. d. Versicherungswissenschaft.*, iv., 1904, S. 275.
- PAUL MASOIN. An Account of the Insane in Belgium, and particularly those in the Colony of Ghel. *Journ. Nerv. and Ment. Dis.*, Sept. 1904, p. 561.
- DUBUISSON. Essai sur la folie au point de vue médico-legal. *Arch. d'anthrop. criminelle*, sept. 15, 1904, p. 677.
- SIEGFRIED TÜRKEL. Die kriminellen Geisteskranken. *Wien. med. Wchnschr.*, Oct. 8, 1904, S. 1926.
- EUGENE S. TALBOT. Criminal Responsibility and Degeneracy. *Brit. Med. Journ.*, Oct. 15, 1904, p. 962.
- CHAS. A. MERCIER. Criminal Responsibility. *Brit. Med. Journ.*, Oct. 15, 1904, p. 957.
- F. J. SMITH. A Suggestion on the Criminal Responsibility of Homicidal and Dangerous Lunatics. *Brit. Med. Journ.*, Sept. 24, 1904, p. 749.

#### TREATMENT—

- DUBOIS. Die Psychoneurosen und ihre psychische Behandlung. A. Francke, Bern, 1904, M. 8.
- DOUMER. Principes fondamentaux de l'électrothérapie des maladies nerveuses. *Journ. de Neurol.*, oct. 5, 1904, pp. 381 et 385.
- CRAMER. Die strafrechtliche Behandlung der Geistig Minderwertigen. *München. med. Wchnschr.*, Oct. 4, 1904, S. 1769.
- J. B. MURPHY. Osmic Acid Injections for Relief of Trifacial Neuralgia. *Journ. Amer. Med. Assoc.*, Oct. 8, 1904, p. 1051.
- EWALD. Gastrische und intestinale Krisen. *Zentralbl. f. d. gesam. Therapie*, Oct. 1904, S. 505.
- The Treatment of Tetanus. Leading Article. *Lancet*, Oct. 15, 1904, p. 1088.
- Surgical.**—A. H. TUBBY and ROBERT JONES. Modern Methods in the Surgery of Paralyzes, with Special Reference to Muscle-grafting, Tendon Transplantation, and Arthrodesis. Macmillan & Co., Ltd., 1904, 10s.
- A. H. TUBBY and R. JONES. Modern Methods in Surgery of Paralyzes. *Clin. Journ.*, Oct. 12, 1904, p. 416.
- HERMKES. Ueber den Werth chirurgischer Behandlung von Neurosen und Psychosen. *Arch. f. Psychiat.*, Bd. xxxix, H. 1, 1904, S. 53.
- VULPIUS. Neurologie und Orthopädie. *München. med. Wchnschr.*, Sept. 27, 1904, S. 1721.
- KRAUSE. Hirnchirurgie. *Deutsche Klinik*, Sept. 1904, S. 953.
- HANS SPITZ. Die Bedeutung der Nervenplastik für die Orthopädie. *Ztschr. f. orthopäed. Chir.*, Bd. xiii, H. 2 u. 3, 1904, S. 326.
- ROBERT KENNEDY. Further Notes on the Treatment of Birth Paralysis of the Upper Extremity by Suture of the Fifth and Sixth Cervical Nerves. *Brit. Med. Journ.*, Oct. 22, 1904, p. 1065.
- MICHAEL HOKE. Study of a Case of Lateral Curvature of the Spine. A Report of an Operation for the Deformity. *Atlanta Journ.-Record of Med.*, Aug. 1904.

# Review of Neurology and Psychiatry

---

## Original Articles

### **SALT STARVATION IN THE TREATMENT OF EPILEPSY**

By WILLIAM ALDREN TURNER, M.D., F.R.C.P.

THE suggestion of substituting the salts of chlorine in the dietary of epileptics by the bromide preparations, is due to Toulouse and Richet (1), who recommended a diet in which the total quantity of sodium chloride per diem was limited to 1 or 2 grammes. It was thought that by diminishing the quantity of the chlorides, the bromides might be administered in smaller doses, and the risk of bromism thereby lessened. The general result of the treatment has been to show that in some cases "salt starvation" has proved a useful adjuvant to bromide medication (Balint, Schaefer (2), and others); while in others little benefit has accrued. The observations of Pierce Clark (3) have shown that the dietary is of especial value in cases requiring large doses of bromides, and in those which show a ready tendency to bromide intoxication. On the other hand, Pandý (4) states that the danger of bromide intoxication is greater, two of his patients having succumbed in this way, and that if it is of any use at all, "salt starvation" merely acts as treatment of symptoms.

As the results of this treatment in the hands of numerous investigators have differed widely, I have thought it advisable to test the treatment in a few cases of confirmed epilepsy. The cases were selected from among the female patients at the Colony

for Epileptics. They had been under observation for a number of years, in consequence of which the severity, character and frequency of their fits were well known. Their mental and physical conditions, and their powers and ability for work were also known. Any temporary fluctuation in the number of the fits, whether from medicinal or dietetic treatment, could in consequence be compared with the natural fluctuations which are common in confirmed epilepsy.

The dietary adopted was Balint's (5) modification of the Toulouse-Richet method. It consisted of milk, butter, eggs, fruit, vegetables and bread. Bromopan, or bread baked with sodium bromide in place of sodium chloride, as used by Balint, was not administered, ordinary household bread being given.

The following is the diet chart :—

*Milk*—1½ litres.

*Pure fresh butter*—50 grammes.

*3 Eggs* without salt.

*Vegetables.*

*Fruit.*

*Household bread*—400 grammes.

*Tea, coffee, or cocoa* with sugar.

The patients were kept at their usual avocations of house and laundry work; and a small dose of potassium bromide, grs. 15 or 30, given at bedtime.

The selected cases were, as far as possible, taken from amongst those whose fits were of considerable frequency. No case of serial epilepsy or of status epilepticus was chosen. All the cases had been at some time on bromides without any benefit.

CASE 1. A. T., æt. 18; a confirmed epileptic of 5 years' duration; both grand and petit mal seizures. Mentally she was slow but worked well. No neuropathic stigmata.

Total Number of Fits.	TREATMENT.		
	4 months before.	4 months of.	3 months after.
	54 fits.	33 fits.	20 fits.

*Pot. brom., gr. 15 omne nocte.*

*General result.*—Some diminution in the number of the seizures during treatment, with continued improvement during

following 3 months. Improvement in the mental condition, so that work was better done. The character of the seizures remained the same. The weight increased 1 lb.

CASE 2. A. J., æt. 19; an epileptic of 12 years' duration; fits of both grand and petit mal variety, the latter largely in excess. Mentally she was dull and slow, with a poor memory.

Neuropathic stigmata were seen in an asymmetrical face and defective lower jaw.

Total Number.	TREATMENT.		
	4 months before.	4 months of.	3 months after.
	81 fits.	69 fits.	82 fits.

*Pot. brom., gr. 30, omne nocte.*

*General result.*—Considerable diminution in the number of seizures during the diet period, to be followed by increase especially of petit mal seizures afterwards. The mental condition also temporarily improved. The patient lost 4 lbs. weight.

CASE 3. F. O., æt. 16; suffered from epileptic fits since 2 years of age; fits of both types, the minor seizures being largely in excess. The administration of chloral had, however, arrested the petit mal attacks, so that the influence of the diet is mainly upon the convulsive seizures. Mentally she was dull and slow, the memory very defective. Neuropathic stigmata chiefly seen in facial asymmetry.

Total Numbers.	TREATMENT.		
	4 months before.	4 months of.	3 months after.
	24 large. 19 small.	29 large. 13 small.	26 large. 23 small.

*Pot. brom., gr. 30, omne nocte.*

The general result, as seen in the total number of seizures, was *nil*. Her mental condition showed some improvement. The effect of the treatment was to eventually increase the number of the seizures. The body weight rose from 6 st. 9 lbs. to 6 st. 11 lbs.

CASE 4. M. B., æt. 36; suffered from epileptic fits since infancy; fits of both grand and petit mal type in about equal proportions. Mentally she was dull, but a fair worker.

Total Number.	TREATMENT.		
	2½ months before.	2½ months of.	2½ months after.
	27	25	21

*Pot. brom., gr. 30, omne nocte.*

The diet produced in this case some general improvement and diminution in the number of the seizures. Her body weight increased from 8 st. 1 lb. to 8 st. 5 lbs.

She has on no previous occasion during the past 5 years had so few fits as during the 2½ months after the above treatment.

CASE 5. F. D., æt. 27 ; an epileptic of 9 years' standing ; suffering from ordinary convulsive seizures. Mentally she was bright, with a fair memory. Neuropathic stigmata were absent.

Total Numbers.	TREATMENT.		
	3 months before.	3 months of.	3 months after.
	22	16	24

*Pot. brom., gr. 30, omne nocte.*

The result *qua* total number of seizures during treatment would seem to indicate some improvement, but it should be pointed out that this patient had previously gone for three months with a total of only 7 fits without any other than bromide treatment. She materially increased in weight after suspension of the diet. The mental condition showed no material change. The body weight increased rapidly after stoppage of the diet.

CASE 6. R. O., æt. 22 ; an epileptic of 17 years' duration ; subject to both grand and petit mal seizures, chiefly the latter. Mentally she was irritable and abusive, had a fair memory, and worked well. Neuropathic stigmata consisted mainly of slight facial asymmetry.

Total Numbers.	TREATMENT.		
	3 months before.	3 months of.	3 months after.
	17 big. 18 small.	16 big. 29 small.	12 big. 16 small.

*Pot. brom., gr. 15* } *nocte manequæ.*  
*Tr. digitalis, m 5* }

The only appreciable result in this case was a clear increase in the frequency of the petit mal seizures. In no previous period of three months had she so many small attacks. The weight remained stationary.

CASE 7. B. L., æt. 25; an epileptic of 11 years' duration; seizures of the ordinary convulsive type. Mentally she was dull and slow, with a poor memory. Neuropathic stigmata were seen in facial asymmetry, a narrow-arched palate, and thin auricular margins.

## TREATMENT.

Total Numbers.	TREATMENT.		
	3 months before.	3 months of.	3 months after.
	28	25	18

*Pot. brom., gr. 30, omne nocte.*

The total number of fits as here recorded would appear to show a steady diminution during and after treatment. It should, however, be stated that in the preceding year, during June, July and August, which correspond to the "three months after" treatment, noted above, the total record of fits was only 10.

There was no appreciable improvement in any other direction. The weight increased 8 lbs.

CASE 8. K. B., æt. 28; an epileptic of 17 years' duration, suffering from both grand and petit mal types, occasionally in series of considerable severity. Mentally dull and slow; facies not characteristically epileptic. She was placed upon the saltless diet, without pot. brom., and the following is the record:—

June,	.	.	.	91 fits.
July,	.	.	.	51 fits.
August,	.	.	.	45 fits.
September,	.	.	.	43 fits.
October,	.	.	.	41 fits.

The diet having proved so satisfactory, this patient is being kept upon it. The mental condition has improved with the diminution in frequency of the seizures. She maintains her weight. This was a case in which bromides were invariably accompanied by increase in the seizures.

*Summary.*—1. In five out of the eight cases a diminution in the total number of fits was observed during the continuance of the diet. It should, however, be remembered in this connection that

confirmed epileptics show great variation and fluctuation in the frequency of their seizures, even when under no treatment at all.

2. In three there was a lessened number of seizures during the three months succeeding the diet; but in one of these it was particularly stated that a more marked diminution had occurred at the corresponding period the previous year, when under no treatment.

3. In three cases the petit mal seizures seemed to have been appreciably increased in number.

4. Although there was some improvement, it cannot be admitted that the influence upon the mental condition was so very marked.

5. The body weight increased in four cases, fell in two, and remained stationary in two.

The *general conclusions* which may be formed as to the advantages to be derived from the administration of the "salt starvation" diet are briefly the following: There are some cases of confirmed epilepsy in which the number of attacks is diminished during the continuance of the treatment, and others in which this improvement has lasted after the diet has been stopped. These are the cases in which the bromides are not well borne, or are even deleterious. I have not seen either the striking improvement amounting to arrest of the seizures described by Balint, or the deleterious influence of salt starvation mentioned by Pandý. The mental condition has shown some improvement, but not to the extent that would warrant the indefinite administration of the diet. This amelioration was no doubt largely dependent upon the removal of the bromides and the diminution in the frequency of the seizures.

The chief disadvantage of the diet is its monotonous character. Patients soon tire of it. As a relief to dyspeptic symptoms it was found to be distinctly useful. It cannot be regarded as in any way a specific remedy in epilepsy, but merely as a relief to symptoms.

#### BIBLIOGRAPHY.

1. Toulouse and Richet. *Acad. des Sciences*, 20, xi.
2. Schaefer. *Neurol. Centralbl.*, 1902, p. 5.
3. Pierce Clark. *Pediatrics*, 1903.
4. Pandý. *Abstr. in Neurol. Centralbl.*, 1903, p. 376.
5. Balint. *Berl. klin. Wochenschr.*, 1901, Nr. 23; *Neurol. Centralbl.*, 1903, p. 347.



**VERTICAL EYE-MOVEMENT TESTING IN THE POSITIONS OF ABDUCTION AND ADDUCTION.**

By LEONARD J. KIDD, M.D.

*Symbols used:* P. = Patient.  
R.S.R. = Right Superior Rectus.  
R.I.R. = Right Inferior Rectus.  
R.S.O. = Right Superior Oblique.  
R.I.O. = Right Inferior Oblique.

WE know that the muscular mechanism of a given movement often varies according to the particular position of the limb, or a part of the limb, during the attempt to perform that movement. Thus the supinated forearm is flexed by the biceps, the pronated or semi-pronated by the brachio-radialis (supinator longus). If we wish to test the biceps we first supinate the extended forearm, and then flex the elbow against resistance. And *vice versa* with the other muscle. If we apply this principle to vertical eye-movement testing we shall find it of great help. The ordinary method is to test these in the so-called position of rest, i.e. with the eyes directed straight forward: the great disadvantage of this is that elevation is effected in this position by the combined action of S.R. and I.O.; and depression by the two depressors acting together. A defect in elevation then shows weakness of one, or both elevators, but does not decide which. We may admit that the recti are primarily and primitively elevators and depressors and became wheel-rotators of the vertical corneal meridian only when the laterally placed eyes migrated to the more nearly central position of man and the higher animals; in like manner the obliques were originally wheel-rotators and became elevators and depressors only later.

We may further admit that a defect of elevation in the position of rest is more likely to show involvement of the S.R. than of I.O.; but further than this we cannot go. Of course we can, and do, clinically test in addition the upward and outward and the downward and outward movements of the eyes. But all the information we gain by these means falls short of that given by the method I shall describe. And first it is essential to remember that in the position of complete abduction of the eye vertical movements of the cornea as a whole are performed

by the two recti, while wheel-rotation of the vertical corneal meridian is performed by the two obliques—in the case of elevation outwards by the I.O., in depression inwards by the S.O. In like manner, in the position of complete adduction of the eye, vertical movements are performed by the obliques, while wheel-rotation is done by the recti—in elevation by the S.R. inwards, in depression by the I.R. outwards.

Now, it is always easier to detect a defect in the movement of the cornea as a whole, than in that of its vertical meridian, though this can often be seen by careful scrutiny of a small patch of iris-pigment either at the top or bottom of the vertical meridian or of a friendly blood-vessel. If we apply all this to the case of apparently isolated ocular palsies we shall see how helpful it becomes.

Let us take the commonest, an apparently isolated palsy of one external rectus, say the right. The R. eye may show marked, or slight, or no appreciable internal squint. We make P. look to his extreme left. We then make him look straight upwards at the rising finger, or pencil, or spill of paper we use. If he does this perfectly we know that his R.I.O. is sound; if badly or not at all, it is affected. A similar attempt to look downwards tells us whether his R.S.O. is affected. We may be able to detect here the inward wheel-rotation by the R.S.R., or the outward by the R.I.R. But here we see that we cannot test the vertical movements of the abducted R. eye owing to the palsy of the R. external rectus. It may be necessary here to resort to the study of the double images, if our examination so far has failed to reveal the presence of wheel-rotation in the position of complete adduction. But we have already proved by our examination that the fourth nerve is not affected, and that the third nerve is either quite unaffected as far as its extrinsic muscles are concerned or else that its palsy is only partial, the R.I.O. being intact.

In like manner we can easily determine, in the case of an apparently isolated palsy of the R. internal rectus, the condition of the two recti, because we can test the completely abducted R. eye in its vertical movements. The wheel-rotation of the obliques is less easy to detect. Let us take now a complete apparently isolated palsy of R. third nerve. If we elevate the dropped lid, P. is looking outwards; he can look to his extreme

R.; we can thus easily test the recti = of course no vertical movement; but we can often see during the attempt to look downwards a slight inward wheel-rotation = integrity of R.S.O. But owing to the inability of P. to look to his left, we cannot test the integrity of the R.S.O. by a study of the downward movement of the adducted eye. (The R.I.O. is of course palsied.)

Finally, in the case of apparently isolated R.S.O. palsy. We tell P. to look to his extreme right, and then to look first downwards and then upwards; we thus test the condition of the R.I.R. and the R.S.R. respectively. The eye, if squinting at all appreciably, is doing so upwards and a little inwards; he can look still more to his left, and then the act of looking still more upwards tells us about the condition of his R.I.O. In this palsy the diplopia, when present (as it usually is), is characteristic and very troublesome; it exists below the horizontal plane, and is shown in such acts as those of going downstairs, counting money, or eating. Of course in any case of real doubt we resort to a study of the double images; and in cases of combined ocular palsies these are often very peculiar. But I have shown, I think, how much the application of the study of the actions of the ocular muscles in the positions of abduction and adduction helps us in the testing of vertical eye-movements by ocular inspection alone.

To sum up therefore:—

- (1) In apparently isolated palsy of external rectus it is easy to test integrity of obliques, less easy of recti.
  - (2) In apparently isolated palsy of internal rectus it is easy to test recti, less easy obliques.
  - (3) In complete apparently isolated third-nerve palsy we can always test integrity of external rectus, and very often of S.O.
  - (4) In apparently isolated S.O. palsy it is easy to test integrity of all muscles supplied by third nerve and that supplied by the sixth.
-

# Abstracts

## ANATOMY.

- A CONTRIBUTION TO THE STUDY OF THE PROTOPLASMIC (410) PROCESSES OF NERVE-CELLS.** S. SOUKHANOFF, F. GIER and GOURÉVITCH, *Névrose*, Vol. vi., 1904, p. 119.

THERE has been much discussion as to whether the collateral processes (gemmules, thorns) of the dendrites, which are so clearly visible by Golgi's chrome-silver impregnation, are artefacts or a natural condition. Ramon y Cajal, Ivanoff and Turner have been able to demonstrate them, however, by methylene blue staining, and the present authors have also succeeded in colouring them by the *intra vitam* injection of the same stain.

It is thus extremely probable that they are actual nervous structures.

GORDON HOLMES.

- THE TERMINAL KNOBS AND THE PERICELLULAR NET. (411)** A. VAN GEHUCHTEN, *Névrose*, Vol. vii., 1904, p. 219.

THE author gives an interesting critical review of the conclusions of Held, Auerbach, Golgi, Donaggio, Bethe, and Cajal, on the mode of termination of axis cylinders round the nerve cells, and on the pericellular nets demonstrated by these workers by their various methods.

His own conclusions were attained from work with Cajal's new neurofibril stain. By it he finds that the knobs by which the axis cylinders terminate round nerve cells or their dendrites are always independent of one another, i.e. do not form a pericellular net, and are merely in contact with the cells. He believes that these terminal knobs (boutons) are identical with the Endfüsse of Held and the Endknöpfchen of Auerbach; and that if a pericellular nervous net, as described by Held, does exist, it consists of homogeneous protoplasm, and that the neurofibrils take no part in its formation, so that it cannot intervene in the function of conduction.

GORDON HOLMES.

- THE NERVE-FIBRES AND THEIR BIFURCATION IN THE (412) SPINAL GANGLIA.** A. MICHOTTI, *Névrose*, Vol. vi., p. 203.

THIS paper includes the results of a study of nerve fibres by Cajal's new fibrillar stain.

The sheath of Schwann is continuous. It appears to be striated. The myeline is transparent. Irregularly arranged

granules are due to the deposition of pigment in the neurokeratin net, but a more regular arrangement of black granules represent the fissures of Lanterman and Schmidt.

The method is a specific stain for axis-cylinders, which are, however, always better impregnated opposite Ranvier's nodes, where it may be so intense that the individual neurofibrils are not visible.

The most interesting part of the paper refers to the relation of the neurofibrils at the point of bifurcation of the single process of the spinal ganglia cells. The question has already been discussed by Cajal, van Gehuchten, Lugaro, and others, whether the impulses of the afferent nerves pass directly into the dorsal roots of the spinal cord or only reach these through the spinal ganglia. This the author seeks to determine from the anatomical relations of the neurofibrils at the point of bifurcation. This bifurcation generally occurs at one of Ranvier's nodes, and the fibrils of both the peripheral nerve and root enter the ganglion, and none are ever visible passing directly from the nerve into the root.

As these are regarded as the essential conductile constituent of the nerves, sensory impulses must pass to the central nervous system through the dorsal spinal ganglia.

GORDON HOLMES.

#### CONTRIBUTION TO OUR KNOWLEDGE OF THE ENDOGENOUS

(413) **SPINAL FIBRES IN MAN.** (Secondary Degenerations in Acute Poliomyelitis.) ROBERT BING, *Arch. f. Psychiat. u. Nerventr.*, Bd. xxxix., H. 1, 1904, p. 74.

THE author gives a careful account of the degenerations found in the central nervous system of a child of four months, who died six weeks after the onset of an acute anterior poliomyelitis. The examination showed a hæmatogenous myelitis in the area of the distribution of the anterior spinal arteries. The grey matter was almost exclusively affected. Larger or smaller areas of this were involved according to the level, the posterior areas being also implicated in the lumbar region.

The following conclusions are drawn from the case:—

(1) The lateral cerebellar tract begins in the region where the second lumbar segment passes into the third. Its longest fibres, that is, those arising from the most distal part of the cord, run in the most dorsal portion of the tract.

(2) The antero-lateral tract of Gowers commences one segment at least below the commencement of the direct cerebellar tract. Its cells of origin lie in the lateral circumference of the anterior horn. Fibres from this pass into the bundle for the most part in

an oblique ascending direction. The longest fibres lie at the periphery of the cord. The lateral cerebellar tract and Gowers' tract terminate in the manner already well determined.

(3) Endogenous fibres are found also in the area of the crossed and uncrossed pyramidal tract. Those in the crossed pyramidal tract originate in the "Strangzellen" in the ventro-lateral part of the posterior horn. For the most part they follow an ascending direction. Some are descending fibres of short course. Those in the direct pyramidal tracts originate in the cells on the median aspect of the circumference of the anterior cornua. Their course is principally ascending, corresponding to Marie's *faisceau sulco-marginal ascendant*; some are descending, corresponding partly to Löwenthal's *faisceau marginal antérieur*.

(4) The endogenous fibres of the antero-lateral root zones, arising from cells in the anterior and posterior cornua, are arranged relatively to each other in such a way that those of longest path are most peripherally situated. In the dorsal part of the lateral columns those are mostly ascending.

(5) The endogenous fibres of the posterior columns originate in the posterior horns. They enter the posterior columns along the median margin of the cornua, and occasionally also along the grey commissure. They are distributed more or less uniformly over the whole area of the posterior columns. The fibres of longest path lie at the posterior periphery, and in the neighbourhood of the median septum. Some of those which rise in the lumbar cord ascend in Goll's column as far as the fourth cervical segment. These endogenous fibres are both ascending and descending, mostly the latter.

ALEXANDER BRUCE.

## THE CORPUS RESTIFORME AND THE BULBO-CEREBELLAR

(414) CONNECTIONS. A. VAN GEHUCHTEN, *Le Névrose*, Vol. vi., 1904, p. 123.

IN this paper the author gives the results he has obtained by the degeneration method and Marchi's stain, on the composition of each corpus restiforme.

He has found that it contains several distinct systems.

1. *Fibræ spino-cerebellares dorsales* (Direct cerebellar tract of Flechsig).—In the medulla this system turns dorsalwards on the lateral surface of the spinal root of the trigeminus, and then bending dorsalwards forms the middle part of the homolateral corpus restiforme. At first these fibres comprise its greatest bulk, but owing to its augmentation by fibres of medullary origin they finally only constitute about a quarter of it.

2. *Fibræ olivo-cerebellares* are only centripetal; they never

degenerate after lesions of the cerebellum or its peduncles. It is difficult to say what proportion of the fibres of this system have their origin in the cells of the olives, as those which come from the *formatio reticularis* and *nucleus lateralis* are also always injured in lesions of the inferior olives. The connection is partly direct, but chiefly crossed. In man they are deep—*fibræ arcuatæ externæ*—in the medulla, but they may be superficial in the lower animals.

3. *Fibræ nucleo-cerebellares*.—This is a homolateral connection of the *nucleus lateralis medullæ* with the vermis, by the way of *fibræ arcuatæ externæ*, which pass through the medial and dorsal portion of the *corpus restiforme*. Russell, Thomas and Probst have described the degeneration of these fibres after lesions of the cerebellum, and the author also found that they degenerate ventrally after section of the *corpus restiforme*, but he regards this degeneration as indirect (retrograde), as it occurs considerably later than the direct Wallerian. The fibres are therefore cerebellopetal.

4. *Fibræ reticulo-cerebellares ventrales*.—The degeneration of these fibres follows disease in the *formatio reticularis medullæ*, independently of lesions of the dorsal nuclei and inferior olives. They are chiefly direct but partly crossed, and as *fibræ arcuatæ externæ* enter the ventral portion of the *corpus restiforme*, bending round the direct cerebellar tract.

5. *Fibræ reticulo-cerebellares dorsales*.—After lesions in the dorsal part of the *formatio reticularis*, chiefly in the neighbourhood of the hypoglossal nuclei, degenerated fibres pass lateralwards through the *vagus nucleus* into the median portion of the *corpus restiforme*. They are both direct and crossed, but in great part direct.

The *fibræ arcuatæ externæ* thus contain fibres of spinal origin, *tractus spino-cerebellaris dorsalis*, and of bulbar origin, *tractus nucleo-cerebellaris*, *tractus reticulo-cerebellaris ventralis*, and probably also part of the *tractus olivo-cerebellaris*.

Further conclusions are: the *corpus restiforme* contains only centripetal fibres; it does not contain direct root fibres; and it has no connection with the dorsal column nuclei, in the rabbit at least, as has been described by the majority of anatomists.

GORDON HOLMES.

# **A CONTRIBUTION TO THE STUDY OF THE OLFACTORY (415) TRACTS. A. VAN GEHUCHTEN, *Nervæze*, Vol. vi., 1904, p. 193.**

GUDDEN described degeneration of the lateral olfactory tract and integrity of the anterior commissure after destruction of the one olfactory bulb. In this he was supported by the work of Loewenthal and others, but Cajal, Probst and Ganser obtained

degeneration in the anterior commissure from the same lesion. V. Gehuchten obtained, however, the same results as Gudden on destroying the olfactory bulb of a rabbit, and concludes that the divergent results were due to a lesion of the olfactory lobes. He regards it as certain that the olfactory portion of the anterior commissure is formed by fibres of the third order, which have their cells of origin in the olfactory lobes.

GORDON HOLMES.

## PHYSIOLOGY.

**MUSCULAR WORK AND THE EXPENDITURE OF ENERGY IN**  
(416) **DYNAMIC CONTRACTION.** A. CHAUVEAU, *Comptes Rendus*,  
June 27, 1904, p. 1669.

THE object of the investigation described by M. Chauveau in this paper is to compare the internal or biologic work of the muscle with the external work which results from its dynamic contraction.

The external work, measured by the product of the weight raised into the height to which it is raised, may increase in two different ways: (1) by an increase in the weight; (2) by an increase in the height to which it is lifted in a unit of time; hence arises a question of practical, no less than scientific importance, namely, how do these two entirely different methods of altering the work affect the expenditure of muscular energy.

Each method was made the object of a series of experiments, the muscles used being those of the forearm. In the first series of experiments five different weights, varying from  $1\frac{1}{2}$  to 6 kilogrammes, were employed, the height and the speed of the movement remaining constant. In the second series of experiments the height and speed of the movement varied, the weight remaining constant. Detailed tables of the results are given, and these are also worked out graphically. The expenditure of muscular energy is measured in terms of the excess of oxygen absorbed during work over that absorbed during rest.

The general result is that in both cases the muscular expenditure increases with the work done, but not at the same rate. The expenditure increases much less rapidly in proportion to the work done in the second case than in the first. It is therefore better in the case of heavy loads to divide the weight and lift each part quickly to the desired height rather than to raise the whole slowly all at once. To give one example. The mechanical result is the same if a weight of 6 kilogrammes is raised to a height of 4.42 metres in 60 seconds, or if four weights of 1.5 kilogrammes are each raised in 15 seconds to the same height. But in the first case



the oxygen absorbed amounts to 325 cubic cm., in the second case to only 243 cubic cm. Thus, if the oxygen absorbed be accepted as the measure of the expenditure of muscular energy, there is in the second case a gain to the muscle of 25 per cent.

MARGARET DRUMMOND.

# **EXPERIMENTS ON THE MOTOR FUNCTION OF THE VAGUS.**

(417) STARCK, *Muench. med. Wochenschr.*, August 23, 1904, p. 1512.

THESE experiments had reference to the relation between the vagus and œsophagus. The writer's method of experimentation was to divide the vagi on one or both sides at various points.

1. Both vagi divided in the neck. The animals soon died through aspiration-pneumonia, the food not being swallowed, the œsophagus paralysed and the cardiac orifice remaining in a light degree of spasm.

2. One vagus divided in the neck, the other immediately below the origin of the recurrent laryngeal nerve. One dog lived twelve days with respiration 8, pulse 160 per minute. It died suddenly while running about, and after death hard fragments of food were found in the œsophagus, which was dilated between the bifurcation of the trachea and the cardiac orifice.

3. Both vagi divided below the parts where the vital fibres are given off, i.e. in the thorax upon the œsophagus. The writer describes the details of the difficult operation by which this was achieved. After five unsuccessful attempts, owing to collapse of the lungs, etc., three dogs recovered completely, none of whom showed either spasm of the cardia, dilatation of the œsophagus, or any interference with swallowing.

4. Both vagi divided immediately above the diaphragm by means of an abdominal incision and traction upon the œsophagus. There was temporary difficulty of swallowing, which he attributes to the operation, followed in every case by complete recovery.

The writer concludes, therefore, that any fibres of the vagus important for the act of swallowing are given off above the root of the lung.

JOHN D. COMRIE.

# **BINOCLAR AND STEREOSCOPIC VISION IN MAN AND OTHER**

(418) VERTEBRATES, WITH THE RELATION TO THE DECUSSATION OF THE OPTIC NERVES, THE OCULAR MOVEMENTS, AND THE PUPIL LIGHT REFLEX. WILFRED HARRIS, *Brain*, Vol. cv., 1904, p. 107.

As a result of experimental lesions of the optic tract in cats, hemianopia is produced with the hemianopic pupil reaction, but

the pupil of the opposite eye is dilated, and reacts more sluggishly to direct light than to consensual stimulation. Lesions of the dorsum of the anterior corpus quadrigeminum in cats produced degeneration in Meynert's fountain decussation, and the author concludes these fibres, or some of them, subserve the pupil light reflex, that Argyll-Robertson pupil is due to a sclerosis of these fibres, and that the consensual contraction of the pupil in man and the higher vertebrates is brought about by a semi-decussation of these posterior pupil reflex fibres, similar to the semi-decussation at the chiasma, and not to any commissural fibres between the sphincter pupillæ centres in the third nuclei. Lesions of the posterior commissure in cats have produced no permanent loss of the pupil reflex to light. Enucleation of one eye, followed by subsequent examination of the chiasmata by Marchi's method in cat, rabbit, pigeon, have shown in the cat semi-decussation of the nerve-fibres, the decussating bundle being considerably the larger; in rabbit only a very few fibres pass into the optic tract of the same side, while in pigeon, owl, toad and chameleon there is total decussation of the optic nerves. The pupils in these animals show a corresponding reaction to light. In the cat there is well marked consensual reaction, while in the rabbit, owl, pigeon and toad only the pupil which is exposed to light contracts. Therefore in these animals there must exist a total decussation of the posterior pupil reflex fibres. A study of the position of the eyes in the head, and of the ocular movements, compared with the decussation of the optic nerves in various animals, show: (1) that the decussation of the optic nerves at the chiasma is complete in all fishes, amphibia, reptiles and birds, whether possessed of binocular vision or not; (2) that binocular vision is originally associated with carnivorous habits, and is found in a few sharks, in some amphibia as the toad, in many carnivorous birds as the larger gulls, the hawks and vultures, owls, nightjar, etc. Amongst mammals binocular vision is especially developed in the carnivora and the primates. In the latter group of monkeys and man the more perfect binocular vision is associated with the development of the hand as a prehensile organ. (3) In animals with laterally placed eyes and periscopic vision the movements of the eyes are independent, as seen typically in the chameleon, while conjugate movements of the eyes for lateral and vertical movements are associated with the acquirement of stereoscopic vision. Convergence of the eyes in the act of feeding is seen in many animals with total decussation of the optic nerves and otherwise independent movement of the eyes, as in the chameleon and hornbill.

The paper is divided into six parts: (1) Binocular vision, and its relation to the decussation of the optic nerves, and to the

feeding habits of the animal. (2) Stereoscopic vision. (3) Macular vision. (4) Ocular movements, conjugate and independent, and their relation to stereoscopic and periscopic vision respectively. (5) The path of the pupil reflex to light, including the results of experimental section of one optic nerve, one optic tract, and of the posterior commissure in cats. (6) Argyll-Robertson pupil. Two plates are given illustrating the position of the eyes in the head in certain sharks and in chameleon, and other woodcuts of sections of various chiasmata, and diagrams of the path of the pupil-light reflex. AUTHOR'S ABSTRACT.

### **PATHOLOGY.**

#### **ON THE ACTION OF VENOMS OF DIFFERENT SPECIES OF (419) POISONOUS SNAKES ON THE NERVOUS SYSTEM.**

GEORGE LAMB and WALTER K. HUNTER. II. A Further Note on Cobra Venom, *Lancet*, Aug. 20, 1904 III. Venom of *Bungarus fasciatus* (Banded Krait), *Lancet*, Oct. 22, 1904.

THE first of these two papers describes the appearance of the nervous system in seven monkeys paralysed with cobra venom. Of these the first four are to be regarded as an addition to a series of six already published (see "Abstract" in *Review*, p. 233, 1904); but they were also examined with special reference to the action of cobra venom on nerve fibres. No unequivocal changes, other-wise than some slight increase of internodal nuclei, could be found in any of the peripheral nerve trunks, even in cases where the degeneration in the ganglion cells was so advanced as to produce a certain amount of vacuolation. The remaining three monkeys were regeneration experiments—that is to say, they were first paralysed by subcutaneous injections of the venom, but subsequently recovered on the administration of an anti-venom. One was killed immediately on the disappearance of all signs of paralysis, and on examination showed degenerative changes in the ganglion cells to be still very apparent. The appearances, however, were rather those of a more chronic degeneration, and in that respect differed from the acute chromatolysis met with in animals dying paralysed with the venom. In the other two monkeys, one killed twenty hours and the other forty-four hours after disappearance of the paralysis, the ganglion cells had completely recovered.

The second paper deals with the appearances of the nervous system in *Bungarus fasciatus* venom intoxication. Seven monkeys were examined, and the time of their deaths varied from fourteen hours to ten days after injection of the venom. In all of them there was a well-marked acute chromatolytic change involving the

motor ganglion cells of cortex, pons, medulla and cord. The type of degeneration was precisely similar to that met with in cobra venom poisoning, but death being so much longer delayed the chromatolytic changes in the ganglion cells were much more advanced than in the cases where cobra venom was used. In those cases, too, where the animal lived several days after receiving the injection an acute muscular atrophy developed, and the condition was therefore that of an acute polio-encephalitis and myelitis, both from the symptomatological and histological points of view. There was little increase in the connective tissue elements round the affected ganglion cells, unless those in the state of disintegration; and the peripheral nerve trunks, even in the monkey living ten days after the injection, showed little, if any, degenerative reaction.

W. K. HUNTER.

**ON THE HISTOLOGICAL CHANGES OCCURRING IN UNUNITED (420) DIVIDED NEEVES.** By ROBERT KENNEDY, *Brit. Med. Journ.*, Sept 24, 1904, p. 729.

THIS paper, read before the Oxford meeting of the British Medical Association, is a defence of the peripheral regeneration theory recently attacked by Mott, Halliburton and Edmunds. Dr Kennedy refers to the process of regeneration according to this theory, and points out that in an ununited nerve the young, immature axis cylinders may be definitely stained by Stroebe's method if the tissue is cut in paraffin so as to insure very thin sections. The regeneration is practically complete in a few weeks, although the final removal of debris by leucocytes, etc., may take a much longer time. He has examined ununited peripheral segments of nerves up to eighteen months after their division, and found the histological characters to be the same as young nerve fibres. To absolutely prevent all possibility of any reunion occurring with other nerve trunks, the author excised a portion of the sciatic of a dog and implanted it in the subcutaneous tissue of its thigh, and then removed it for examination in six months' time. Mott, Halliburton, Edmunds, and also Langley and Anderson, would explain the existence of axis cylinders in such an ununited nerve by claiming that as small nerve twigs must be severed at the time of planting the graft, these fibres may penetrate into the portion of nerve. Surely, however, if there was such an affinity for nerve fibres to seek out nerve tissue in a graft, there would be no reason for aiding Nature in repairing a divided nerve. But secondary suture has in many cases to be performed, a fact which throws much doubt on the explanations given by these authors.

Kennedy criticises the stress laid by Mott, Halliburton and Edmunds on the necessity for the presence of excitability and conductivity as the definite proof of a distal segment containing young nerve fibres, and argues that there may be a stage when no such conductivity is demonstrable and yet immature nerve fibres may be in existence. He also refers to the want of credence which Messrs Mott, Halliburton and Edmunds show towards the well-known clinical fact that after secondary suture, sensation may return in the affected area of skin within two days. He cannot accept the theory that this may depend on the freshening of the central end of the divided nerve. Dr Kennedy explains the care with which the clinical fact of returned conductivity of sensory impulses is or should be ascertained. With the eyes covered a needle is inserted into the affected skin. The rule is that after one or two days the patient feels pain when this is done. This is repeated day by day until complete restoration to the normal, and records are kept of localisation of sensation and thermic sensation. He has about forty cases from which he derives these data. Mere recognition of touch, which might depend on neighbouring sound nerves, is not taken as a proof of restoration of the conduction of sensation.

Dr Kennedy refers to the appearances in the distal portion of the central end of a divided nerve, and refers to the large number of small fibres present, which he thinks are young fibres regenerated in a similar way to those in the peripheral end.

ROBERT A. FLEMING.

**EXPERIMENTAL SECONDARY DEGENERATIONS STUDIED BY  
(421) THE METHOD OF DONAGGIO. LUGIATO, *Riv. Sper. di Fren.*,  
May 1904.**

THE author has published a preliminary note on the results which he has obtained by Donaggio's method for primary degenerations, after tearing out the sciatic nerve.

The animals operated on were killed at varying periods after the operation, and the interval which was necessary after the operation for a degeneration to appear was determined.

It was found that if the animal was killed twenty-four hours after the operation was performed, there was no evidence of any degeneration by Donaggio's method, and of course there was no reaction with Marchi's method. At the end of the second day, however, there was abundant evidence of degeneration in the lumbar region in Burdach's column, which could be traced into Goll's column higher up. Some degeneration of the anterior root-fibres, which corresponded to the injured roots, could be traced to

the anterior cornua. The Marchi method again gave negative results. It will be seen, therefore, that the method of Donaggio can reveal a lesion at a phase which is not demonstrated by the method of Marchi.

On the third day, however, slight evidences of degeneration are visible by the Marchi method, and the lesion is plainly manifest by this method on the fourth day.

The author has also found that the lesion which was shown by Donaggio's method on the second day has much increased by the fifth day. It will be seen, therefore, that the fibres, which will degenerate as the result of the injury, do not all degenerate at the same time or the same rate, and he hopes to give the chronological order of the lesions of the various fibres which degenerate in the same column, in a future paper.

In one or two cases the author has found that following a tearing out of the sciatic nerve on one side, there has been a bilateral lesion in Goll's column. At present this is difficult to explain. We shall await the further results of the experiments with great interest.

R. G. Rows.

**POSITIVE COLORATION OF NERVE FIBRES OF THE CENTRAL  
(422) NERVOUS SYSTEM IN THE INITIAL STAGE OF PRIMARY  
AND SECONDARY DEGENERATION. A. DONAGGIO, *Ris.*  
*Sper. di Fren.*, May 1904.**

THE author, referring to the work of Vassale and others, compares the primary degeneration of nerve fibres, which is characterised by a slow atrophy of the myeline sheath without destruction of the axis cylinder, at least for a long time, and which is produced by toxines, with secondary degeneration which is produced by an interruption of the fibre, including the axis cylinder. In this last a complete disintegration of the whole fibre takes place, in fact this represents the initial phase of the process. This is followed by a second phase, which consists of a hypertrophy of the interstitial tissue.

A partial phase of hypertrophy of this tissue is seen in a bundle injured in a primary way, when the toxin is so virulent as to produce an interruption of the whole fibre.

The difference between the primary and secondary degeneration is shown by the difference of reaction with osmic acid.

The fibres which are undergoing secondary degeneration are coloured black in the earlier stages, and when the regressive products are removed, the proliferation is shown by Weigert's method.

But the fibres which are undergoing primary degeneration give a negative result with the Marchi method; they may, however,

also pass through one of two phases. They may either recover, if the toxic agent is removed, or they may go on to an advanced degree of atrophy, and be replaced by a hyperplasia of the interstitial tissue, if the action of the toxin is prolonged. Up to this time, however, we have not been able to obtain a positive coloration of primary degeneration. But, as the author notes, these primary degenerations are very common, and he suggests that many conditions which have led to the use of the term "functional lesions" may be due to such primary degenerations, which are not demonstrable by our present technique.

In order to supply this deficiency, the author has conducted a series of experiments, and he has arrived at the following result: that nerve fibres in the first phase of primary degeneration and also in the earliest phase of secondary degeneration, if fixed with bichromate of potassium and coloured with hæmatoxylin, and then exposed to the action of various metallic salts, *e.g.* those of tin, copper, or iron, acquire the power to resist the processes of decoloration more than the normal fibres. Decoloration is carried out by Pal's method.

The author has used three salts—ammoniated chloride of tin, neutral acetate of copper, and perchloride of iron. With the first the myeline sheath and the axis cylinder are both stained violet-red, but the structure of the two can still be distinguished. With the second the whole fibre is stained black, or it may be grey, or it may be red. With the third the fibres are stained black.

The author draws attention to the fact that in cases of mental disease he has been able to determine lesions, in which the fibres were in the first stage of primary degeneration, but which could not have been demonstrated by ordinary methods.

R. G. Rows.

# **STUDY OF THE OPTIC NERVE IN TABETIC AMAUROSIS.**

(423) LÉRI, *Nouv. Icon. de la Salpêtrière*, Sept-Oct. 1904, p. 358.

THE richness of the material at the author's disposal, and its adequate handling, combine to render this contribution to a somewhat neglected subject an important and valuable one. An exhaustive examination of the optic nerves was made in 21 cases of complete, and 2 cases of incomplete, tabetic amaurosis, and in 3 cases of general paralysis with blindness. As a control, the corresponding parts in 16 non-amaurotic tabetics and 18 general paralytics were taken. In addition, the optic nerves from 6 syphilitics with medullary lesions other than tabes, from 2 cases of unilateral optic atrophy, from several apparently normal individuals, and from other cases of which it is unnecessary here

to specify the details, were sectioned. In all, 84 cases were included in the research. The article is accompanied with numerous photo-micrographs and drawings.

What strikes one on examination is the practically invariable thickening of the meninges, and at the same time the remarkable variability in size of nerves apparently equally deprived of sound fibres. In the majority of cases of tabetic amaurosis, the optic nerves are much diminished in size throughout their extent: they are often flattened, ribbon-like sometimes, scarcely more than 1 mm. in diameter. Nevertheless, in other cases, the nerve may be void of sound myelinated fibres, and yet maintain its volume, filling the meningeal sheath as if normal. There must be, therefore, some process other than a simple atrophy at work.

Detailed microscopical examination shows that the dural sheath is seldom altered; it may be a little thickened. The arachnoid is usually detached, and the subarachnoid space increased, and almost always filled with an abundant infiltration of round cells undeniably lymphocytes. They fill the intervaginal space and invade the thickened pia, sometimes forming therein veritable nodules. They show a proclivity for development in the neighbourhood of blood-vessels.

As far as the optic nerve itself is concerned, two types may be distinguished, the large and the small. The latter occur with much greater frequency; on transverse section they are made up of a homogeneous granular tissue, sometimes finely vacuolated, scattered throughout is an infinite number of round or oval nodules or nodes, each of which is formed by concentric layers of fibrous tissue; not infrequently one or two red blood corpuscles remain in the centre.

In the granular tissue will usually be found one or two isolated nerve fibres.

In the large type of tabetic atrophic optic nerve, fibrous trabeculae unite the nodules, thick dense sclerotic fibrous trabeculae which have strangled the nerve fibres.

The examination of the process at an earlier stage would make it seem that the nodules are found at the "nodal points" of the normal interfascicular connective tissue. And further, if it be remembered that in the normal nerve the blood-vessels are found in relation to the interfascicular connective tissue, whereas in the neuroglia are only lymph spaces, it will be seen that the author's contention that the primary lesion is an intense vasculo-conjunctive or conjunctivo-vascular new formation is far from improbable. Apparently, then, in an advanced stage of the condition we find superadded to the first process, the vascular new growth, a secondary process of defence, a tissue reaction indicated by intense sclerosis.



To use other words, throughout the whole optic nerve the lesion of tabetic amaurosis is an interstitial neuritis, a syphilitic cirrhosis of vascular origin, plus a syphilitic meningitis.

There is a close relation between these anatomo-pathological findings and the clinical features of tabetic amaurosis. For clinically two periods may be distinguished: the first of very short duration, characterised by rapid loss of vision, frontal headaches, phosphenes, visual hallucinations, and with the ophthalmoscope, by an irregular disc, pale grey or white, with pigmentary deposition, apparently the index of a more or less marked neuritis; the second much longer in duration, during which luminous sensations are conserved, the disc being distinctly white or grey against the red background of the retina. Pathologically we have a first stage of optic and meningeal irritation, lymphocytosis and vascular new formation, a second stage of arrest, and indeed of reparation rather than evolution.

The significance and bearing of this article on current theories of the "primary" optic atrophy of tabes will readily be appreciated.

S. A. K. WILSON.

**THE PATHOLOGICAL HISTOLOGY AND PATHOGENY OF**  
(424) **SYRINGOMYELIA.** THOMAS and HAUSER, *Nouv. Icon. de la Salpêtrière*, Sept.-Oct. 1904, p. 376.

THE present paper is not the least important of recent serious contributions to a still vexed question, the pathological histology and pathogeny of syringomyelia. It is an elaboration of a previous article on the "Pathogeny of Certain Medullary Cavities" (*Revue Neurologique*, 30th Oct. 1902), in which the conclusion is arrived at that cavity formation in the cord from loss of tissue substance may arise primarily from vascular lesions, and that these same cavities may become ultimately lined and invaded by neuroglia from their walls under the influence of a secondary inflammatory reaction. The same process is advanced as a cause (if not the cause) of syringomyelic cavitation of the cord.

A single case of the disease is taken and analysed and exhaustively examined. The weak point in the research is the insufficient material for the theory. It is true that one or two other syringomyelic cords were examined, but that can scarcely be regarded as affording conclusive evidence for a pathogenic hypothesis for so complex and variable a condition as syringomyelia.

To be brief: the fundamental lesion in the authors' case is a conjunctivo-vascular one; it is chiefly composed of adult fibrous connective tissue, plus an abundant vascular new formation. Everywhere throughout the cord were sinuous tracts of wavy

homogeneous vascular connective tissue, manifesting a tendency to vegetative overgrowth; the invasion of the outer layers by neuroglial fibres gives the papilliform appearance so common in syringomyelic cavities. The luxuriant fibrous formation seems to be a direct derivative from the adventitia of the blood-vessels; in other words, originally perivascular, it may become independent. Round the lesion, what of the nervous parenchyma? Usually there is a more or less intense neuroglial reaction, and in this zone the nerve fibrils are found to be innocent of myelin sheaths; the axons are probably injured too, but of this the authors are less certain. Once the primary lesion diminishes in intensity, the neuroglial reaction ceases too.

One cannot but be struck by the fact that the topography of the initial lesion recalls the spinal distribution of certain arterial systems. The central artery and the arterioles of the posterior horns of grey matter are in the line of the lesion; in the white matter it often is found along the course of the artery or arteriole of the posterior median septum.

The fissures and actual cavities are no doubt to be attributed to the disappearance of minute *sequestra* of nervous parenchyma, minute islets separated and killed by the advance of the vegetative process.

The general discussion which forms the latter part of the article is largely historical, but various cases are taken to show that the hypothesis advanced will account for a considerable number of the facts of syringomyelia.

A word must be said in regard to the altogether admirable drawings which accompany the paper. In execution and in lucidity they leave nothing to be desired, and for the purpose of the authors they are infinitely superior to photo-micrographs.

S. A. K. WILSON.

## CLINICAL NEUROLOGY.

### PERINEAL ZOSTER, WITH NOTES UPON CUTANEOUS (425) SEGMENTATION POSTAXIAL TO THE LOWER LIMB.

HARVEY CUSHING, *Amer. Journ. of the Med. Sci.*, March 1904.

DR CUSHING has extirpated the Gasserian ganglion in twenty cases. In two of these, within a few days of the operation, an herpetic eruption appeared on the opposite side of the face. In the first case a few herpetic blisters formed on the upper lip and ala of the nose of the left (sound) side on the third day after the operation. On the following day a crop of newly formed and forming vesicles were observed in the region of the perineum confined to the right side, and most numerous about the margin of the anus. It was

also possible to delineate a cutaneous area of actual tactile and thermic hyperæsthesia, which was nevertheless hyperæsthetic to painful stimuli, pressure, pinching, pricking or scratching over the lower portion of the right half of the scrotum, the posterior surface of the buttock and posterior aspect of the right lower limb to about the middle of the calf. By the twenty-eighth day after the operation some diminution of thermic sensation was the only defect over this area, and the anal and cremasteric reflexes which had disappeared were again present.

The herpetic eruption in the second case appeared on the seventh day after the operation, the patient having exposed himself two days previously to a chill. The vesicles were distributed over the second division of the trigeminal nerve on the left (sound) side, also on the neck overlapping the angle of the jaw. On the right side there was a symmetrically placed patch near the angle of the jaw entirely outside the post-operative area of anaesthesia. In addition there was a symmetrical area of hyperæsthesia to painful stimuli and hyperæsthesia to tactile and thermic stimuli, including the margin of the anus, perineum and lower portions of the scrotum; no vesicles appeared over this area, however.

The occurrence of bilateral zoster affecting symmetrical segments is practically unknown, hence, as the author remarks, "many will consider the facial eruption in these two cases to have been of the nature of *H. febrilis seu labialis*, which is so commonly bilateral." But the circumstance that no vesicles developed in the anaesthetic area resulting from removal of the ganglion, makes it probable that the presence of the ganglion is essential for the production of the herpes.

"It would seem that gravitation must have played a part in bringing about a deposition of the infective agent, whatever it may have been, in the caudal portion of the meningeal sac and the anatomical position of the terminal sacral ganglia at the very lowermost part of the sac would perhaps favour such a view."

The author discusses sensory segmentation postaxial to the lower limb, and the methods by which information regarding it is derived, illustrating the views of Starr, Thorburn, Head, Kocher and Wichman as to the distribution of the several sacral areas.

EDWIN BRAMWELL.

**A FATAL CASE OF TABETIC GASTRIC CRISIS.** LEONARD J. KIDD, (426) *Lancet*, Nov. 5, 1904, p. 1282.

A MALE, who had acquired syphilis at the age of twenty-five, began nineteen years later to suffer from severe attacks of vomiting, lasting for one or two weeks, and unaccompanied by pain. The

patient had been seen by several neurologists, all of whom had diagnosed his case as tabes. The author saw the patient during one of these attacks three years after their commencement. The vomiting had then lasted for six days, and showed no signs of yielding. The patient died of prostration on the twelfth day of the attack. Author advises resort to morphine, which was not used in this case, before marked prostration present, also care in diet of tabetics.

AUTHOR'S ABSTRACT.

**A CLINICAL LECTURE ON REMISSIONS AND RELAPSES IN**  
(427) **INSULAR SCLEROSIS.** T. BUZZARD, *Lancet*, July 16, 1904.

NOTES are given of cases which illustrate well the frequent occurrence of remissions or recoveries and relapses as a feature of insular sclerosis. This peculiarity of the disease has frequently led to an erroneous diagnosis of hysteria, and Buzzard thinks that "the common picture of hysteria has been somewhat largely based upon a misinterpretation of such symptoms."

The remarkable tendency to the occurrence of remissions and relapses may also throw some light on the ætiology of the disease. Buzzard, as is well known, advocates the view that insular sclerosis is of purely toxic origin—due to "the presence in the blood-vessels of an infective agent which sets up an inflammatory process in the interstitial tissue." One argument in favour of this view, and against the endogenous (congenital) origin of the disease, is found in the tendency to recoveries and relapses. Nervous diseases of endogenous origin are usually characterised by their comparatively steady progress, while it is well known that in one disease, pre-eminently of infective origin, viz., syphilis, the tendency to recovery and relapse is often very evident. Other facts, pointing in the same direction, are the grossness of the anatomical lesions in insular sclerosis, its very infrequent occurrence as a family disease, and the remarkable variations in the degree of acuteness of the course of the disease.

Redlich's observations, that cases of insular sclerosis may show profoundly affected nerve fibres with relatively little affection of the neuroglia, and that vascular changes occur only in a certain number of cases, are not regarded as conclusive evidence against the inflammatory origin of the disease. The typical parenchymatous degeneration of tabes may be accompanied by gross intracranial endarteritis. "Presumably, therefore, a toxin may give rise coincidently to each form, and in some cases of insular sclerosis—or in some localities in a particular case—its influence may fall upon the nerve fibres rather than on the vessels and interstitial tissue."

A. W. MACKINTOSH.

**INTRA-MEDULLARY ABSCESS OF THE SPINAL CORD.** An  
(428) Account of Three Cases. ALDREN TURNER and JAMES COLLIER,  
*Brain*, 1904, Part cvi. p. 199.

THE cases described were examples of pressure or evascularisation paraplegia resulting from carcinoma, pachymeningitis and spinal caries respectively. The area of pressure-myelitis resulting had become secondarily infected with the common organisms of sup-puration, the source of infection being presumably the bladder, which was in each case the seat of a severe cystitis.

In the area of the pressure-myelitis, pus was present in the interstices of the spongy necrotic tissue, but above and below this area the pus had spread widely in the form of a very definite abscess cavity, with well-defined wall, and situated in the planes of least resistance similarly as are the cavities in syringomyelia and as are the extravasations in hæmatomyelia.

The longitudinal extent of the abscess cavity was considerable in each of the cases, and in one of them it reached from the fourth cervical segment to the conus medullaris. There was evidence that the abscess formation extended in the spinal cord chiefly by cleavage of the tissues along the lines of less resistance and also to a less extent by tissue involvement. In two of the cases there was little or no clinical evidence of the presence of abscess, and it seemed probable that the abscess formation had occurred and had extended rapidly during the period of profound physical prostration which preceded death. In the third case high pyrexia and a rapid upward extension of the spinal signs were indicative of the presence and extension of abscess.

Thirty-five illustrations from photographs and projection drawings of sections of the spinal cords in the three cases are figured.

The cases are interesting on account of their variety, and the authors at the time of writing had been unable to find any similar cases recorded, but their attention has since been drawn to several recent publications describing precisely similar cases, the most inclusive of which is that of Andre Léri from M. Marie's laboratory at the Bicêtre.

In some of these cases the abscess formation has been described as part of the process of primary myelitis.

**AUTHORS' ABSTRACT.**

**A CLINICAL REPORT OF THREE CASES OF INJURY TO THE**  
(429) **LOWER SPINAL CORD AND CAUDA EQUINA.** THEODORE  
H. WEISENBERG, *Am. Journ. of the Med. Sci.*, May 1904.

THE first patient presented bilateral peroneal palsy and steppage gait, the last lumbar and first two sacral segments being those probably affected.

The second case was one of bullet wound, in which the lesion implicated the cauda equina; the knee-jerks were increased.

The third case had "a form of Brown-Séquard paralysis peculiar to cases of unilateral lesions in the lower end of the cord," motion and sensation were affected in the same lower limb, while sensation of the external genital organs and perineum of the opposite side was also disturbed.

EDWIN BRAMWELL.

**A CONTRIBUTION TO THE STUDY OF THE CORTICAL LOCALISATION OF VISION. A CASE OF QUADRANTIC HEMI-ANOPIA WITH PATHOLOGICAL EXAMINATION.** CHARLES BEEVOR and JAMES COLLIER, *Brain*, 1904, Part cvi. p. 153.

THE case reported was that of a man, aged 55 years, who had suddenly developed a left upper quadrantic hemianopia, which persisted as the sole physical sign of local cerebral lesion until his death, which occurred twenty months later.

The blind field was exactly limited to the left upper quadrant, the fixation spot escaping. The lesion was due to thrombosis of certain branches of the posterior calcarine artery. It was confined to the cortex, and it involved (1) the right fusiform lobe for its posterior 2·5 cm.; (2) the right lingual lobe from the junction of the calcarine and parieto-occipital fissures to the pole of the hemisphere, the vertical polar gyrus being intact; (3) the whole of the cortex of the depth of the calcarine fissure; (4) the greater part of the inferior cuneal gyrus above the calcarine fissure, a small area only at the anterior and posterior limits of the gyrus being free. The necrosis did not involve the optic radiation at any point. The anatomy is illustrated by two plates and a perimetric chart is figured.

The case is of great importance, as there is only one other case of pure quadrantic hemianopia from a cortical lesion on record which has been examined pathologically (that of Hun). The authors conclude that the area of the visual cortex is co-extensive with the presence of Gennari's stripe, and that it is not narrowly limited to the cortex of the lips and depth of the calcarine fissure, but extends over the whole cuneus and lingual lobe, the separation of the upper and lower fields being only, roughly speaking, the line of the calcarine fissure.

They are of opinion that compensation occurs in the cortex, limiting a partial lesion of the half vision centre.

AUTHORS' ABSTRACT.

**INFANTILE CEREBRAL HEMIPLEGIA AND HEMIATAXIA.**  
(431) BOUGHAUD (de Lille), *Rev. Neurolog.*, Oct. 15, 1904, p. 996.

IN this paper is described the case of a child with an uneventful family and personal history, who at the age of four and a half had an attack of right-sided weakness without any acute symptoms—fever, convulsions, etc.—at the onset, but accompanied with the presence of curious movements which only appeared during voluntary movement, and were characterised by ataxy and inco-ordination. These movements affected the leg as well as the arm, and in the face there were at first spasmodic twitching movements. The onset of the illness was characterised by languor, sleepiness, and mental dulness. As time went on, the weakness of the limbs became less marked, the twitching of the face disappeared, the leg improved very much, and no contracture developed. But the upper limb remained affected with the ataxy and inco-ordination when any voluntary movement was attempted.

The author enumerates and discusses the various forms of hemiplegia and diplegia occurring in early life, and concludes that his case is an exceptional one. He also gives particulars of another case with similar inco-ordinate movements, accompanying a hemiplegia dating from birth, ushered in with convulsions, and presumably the result of a lesion, arising during a long and difficult labour.

The occurrence of the same clinical symptoms in two patients in whom the onset occurred under such different conditions, would suggest the hypothesis that the unusual symptoms are related to, not the nature of the initial lesion, but its situation. Such cases are much more uncommon than the cases in which athetoid movements are present, but the writer has seen two in which the determining condition was an attack of infantile hemiplegia attended at the onset with fever and unilateral convulsions.

JAMES TAYLOR.

**THE DIAGNOSTIC VALUE OF HEMIPLEGIA OF GRADUAL**  
(432) **ONSET AS A SIGN OF CEREBRAL TUMOUR.** R. T.  
WILLIAMSON, *Practitioner*, Sept. 1904, p. 321.

THE author instances three cases of cerebral tumour with progressive hemiplegia, and in which no optic neuritis was present. His conclusion is summed up as follows:—"Provided there are no indications of cerebral abscess, progressive hemiplegia or hemiplegia of gradual onset, in which weeks or a few months elapse before the paralysis is complete, may be regarded as strong

evidence of cerebral tumour, even when optic neuritis is absent, or when optic neuritis, headache and vomiting are all absent."

STANLEY BARNES.

**ON THE SYMPTOMATOLOGY OF PARALYSIS AGITANS. L.**  
(433) BRUNS, *Neurol. Centralbl.*, Nov. 1, 1904, p. 978.

THE author draws attention to a number of the rarer symptoms of paralysis agitans which he has met with in the clinical examination of 74 cases. He points out that often the patient was seen on one occasion only, and hence these notes do not give reliable statistics as to the frequency of the symptoms which he describes.

Salivation was present in five cases, and Bruns believes that it is due to a real primary hypersecretion of saliva, owing to bulbar pathological changes, and not to difficulty in swallowing the saliva, as has been suggested by some writers. In two of his cases salivation was a very early symptom, and occurred at a period when there were no signs of tremor, paresis or rigidity of the muscles supplied by the nerves arising from the medulla and pons. In three other cases, however, salivation occurred only at a late period, and marked symptoms of bulbar paresis were then present. In another case there were severe bulbar symptoms, but salivation was not present.

Four cases are recorded in detail, in which there were disturbances in the functions of the muscles supplied by the bulbar nerves. These symptoms were difficulty in swallowing, defective movements of the muscles of the lips, tongue and soft palate, and of the muscles of mastication. In three cases there was also a speech affection of a dysarthritic character. In these cases the disturbances in the functions of the bulbar muscles are due to paresis, associated with rigidity and slowness of movements, similar to the affection of the muscles of the trunk and extremities in advanced cases of paralysis agitans.

Excessive sweating was a symptom in two cases. A subjective feeling of heat was present in many cases. In two cases there was atrophy of the interossei muscles of the hand, and the skin of the fingers was thick and glossy. In these cases, according to the patients, the hands and feet had recently increased in size, and the nails had become flat and short.

In another case the hands had increased in size, but the changes were due chiefly to increase of the soft parts. The reflexes were usually normal in the cases seen by Bruns. In one case there was dragging of the foot, which was in the position of talipes equinus.

Pain, of a rheumatic or neuralgic character, is mentioned in all



descriptions of the disease. In seven cases seen by Bruns it was very severe; mostly it occurred at an early period in a limb which was affected with tremor at a later date.

In one case Bruns found the typical symptoms of paralysis agitans associated with bilateral progressive nuclear ophthalmoplegia—unilateral ptosis, bilateral paralysis of the external rectus, and other symptoms of oculomotor paralysis. The ophthalmoplegia had preceded the symptoms of paralysis agitans. In another case there was unilateral ptosis. In a third case, nystagmus was present. Whilst the tremor of paralysis agitans very often ceases entirely on active movement, there are other cases (not so rare, according to Bruns) in which the tremor of the arm occurs during active movement. This condition was observed in eight cases: it was noted chiefly at an early period of the disease, and in cases in which the symptoms were unilateral. Usually in such cases there was slight tremor also during repose; but this tremor was increased by voluntary movement. In these cases the tremulous movements differed from those of disseminated sclerosis by being of uniform amplitude. A case is briefly recorded in which there was great difficulty in diagnosing between paralysis agitans and disseminated sclerosis, on account of the tremor simulating that of the former disease during rest, and that of the latter during movement.

In one case Bruns has observed the combination of tabes with paralysis agitans.

R. T. WILLIAMSON.

**A CONTRIBUTION TO MYASTHENIA GRAVIS.** H. OPPENHEIM,  
(434) *Deutsch. med. Wchschr.*, No. 29, 1904.

THE case described is that of a cutler, aged 47, who came to the ophthalmic clinique complaining of double vision, and was found to have paralysis of both internal recti and crossed diplopia. The diplopia had developed suddenly after a chill three months previously, and for a day or two at that time the patient had had some headache and giddiness, which were not, however, severe. When seen by the author there was marked bilateral ptosis of recent development which was said to vary greatly. The levatores palpebrarum were very easily fatigued, and the ptosis was increased by exercising other muscles. The probability that this was a case of ocular myasthenia gravis was corroborated by the patient's statement that the ptosis and diplopia were absent for some hours after waking in the morning, and by the demonstration of the myasthenic reaction in the left deltoid muscle. Although this muscle could not be completely exhausted by the interrupted or

continuous current, the contractions became obviously weaker. In addition to the above, a developmental anomaly of the right foot (a rudimentary digit) was present. In other respects the examination of the nervous system and internal organs was completely negative; motion sensation, the reflexes, etc., being normal.

The author remarks that these ocular forms of myasthenia gravis are not generally recognised, and that they are apt to be mistaken for cerebral syphilis or tabes. In myasthenia gravis, as in the latter affection, the appearance of the ocular paralysis is usually unaccompanied by headache or vomiting. Bilateral ptosis dependent upon hypotonia of the levator palpebræ is not uncommon in tabes, and in this disease the ocular paralyses not unfrequently vary in intensity from time to time. The pupil signs of tabes are not found in myasthenia gravis, but in rare cases they may be absent in the former affection. Where this is the case, and the diagnosis is in doubt, the muscles should be examined for the myasthenic reaction which may be present, as in this case, in muscles which show no defect in function. Oppenheim has in previous writings pointed out the comparative frequency of congenital anomalies among reported cases and he holds the view that the disease is of congenital origin.

EDWIN BRAMWELL.

**REMARKS ON SOME TICS OF CHILDHOOD.** MEIGE and (435) FEINDEL, *Rev. de Neurol.*, Sept. 20, 1904.

FOUR cases are recorded in this paper:—1. Girl, aged eight, who had the following tics, of one month's duration—wrinkling up the nose, winking, spasmodic movements of the eyeballs, and frowning. She had, moreover, the habit of biting her lips, as well as an occasional stammer. In this mild case the treatment was (1) to correct the grimacing, (2) to allow a good deal of rest in bed, and (3) to withdraw from school. 2. Girl, aged eight and a half, who for eighteen months had suffered from a tic consisting in nodding the head; thereafter she developed a spasmodic cough, and a habit of jerking her elbows in towards her sides and at the same time giving an expiratory grunt. Sometimes instead of this gesture the forced expiration was preceded by slapping the epigastrium. She also had the habit of grimacing, of biting her lips, of restlessness, and of eating too quickly. Probably the tic of the arms originated in an attack of abdominal pain or discomfort. The treatment was rest in bed for a fortnight, correction of the habits, the prohibition of heavy or unstable head gear (a fertile cause of nodding tics) and the use of quinine ointment for the lips, with respiratory gymnastics for the grunting. 3. A girl of thirteen

with multiple tonic and clonic tics—eyes, mouth, nose, forehead, laryngeal muscles, shoulders and arms. She had also certain “phobias,” and habits of repetition of words or gestures. In this case the malady was principally due to faulty upbringing, and treatment consisted in withdrawing her from home influences. 4. Boy, aged sixteen, who had, from his thirteenth year, suffered from tics of the facial muscles and nodding movements. He had also choreiform spasms of the fingers, and in walking knocked his heels together. He also had the habit of frequently stooping down to pull at his stockings. He presented some of the stigmata of degeneration; was of low intelligence, sullen, passionate and impulsive. In his case isolation and rigorous discipline are required, and the prognosis is less favourable than in the preceding.

J. S. FOWLER.

**BILATERAL SPASM OF THE MUSCLES OF THE NECK AND**  
(436) **FACE** GAUSSEL, *Nouv. Icon. de la Salpêtrière*, Sept.-Oct. 1904, p. 337.

A YOUNG soldier, aged 23, of an emotional, impressionable and sensitive nature, was seized suddenly during drill with a spasm of his sterno-mastoids, a condition which returned whenever commanded to direct the head to one side or the other. After a year his entrance into hospital allowed of a careful examination, which revealed a marked forward inclination of the head, the chin touching the sternum, a “triple chin,” a palpable bilateral contracture of the platysma and of the sterno-mastoids. When a spasm supervened, the facial groups were usually involved, the *alæ nasi* dilated and contracted, the forehead wrinkled, the lips twitched, and a blepharospasm likewise occurred.

The crises come and go at irregular intervals, their duration is equally irregular. This symptom complex apart, the patient is in excellent health.

Undoubtedly the condition is to be grouped as a tic, though not to be explained as professional, or of habitual physiological function; the muscles affected are innervated from two different sources. All therapeutic attempts have proved eminently unsatisfactory.

S. A. K. WILSON.

**THE CONDITIONS UNDER WHICH BABINSKI'S SIGN MAY**  
(437) **APPEAR IN EXTRA-PYRAMIDAL LESIONS.** BERTOLOTI,  
*Riv. d. Pat. nerv. e. ment.*, Sett. 1904, p. 430.

OCCASIONALLY cases have been recorded in which Babinski's phenomenon was present in a typical form in extra-pyramidal

lesions. Among these may be instanced one or two cases of peripheral neuritis (Crocq, Lortat, Jacob) and of infantile paralysis (Boeri). The author reports two cases in this category. The first concerned a young man of 20, with a lesion involving the second and third sacral roots—more on one side than the other—the result of spinal caries. In addition to severe root pains, paralysis and atrophy in part of the flexor groups of the lower limbs, presence of the knee-jerk and absence of the Achilles-jerk, the patient presented a characteristic extensor response on both sides, and also a double Strümpell's tibialis phenomenon. There was a complete reaction of degeneration on the right, incomplete on the left, in the muscular groups supplied by the internal popliteal and posterior tibial nerves. The second was a case of poliomyelitis in a child of 5, with atrophy of the left lower extremity, the reaction of degeneration being found in all the muscles supplied by the internal popliteal nerve. On the left, stroking the sole gave an extensor response, the right being flexor.

The two cases are obviously alike in that there was an alteration of the normal equilibrium between the flexors and extensors of the toes. A general discussion on cutaneous reflexes follows. The writer believes that so-called pathological cutaneous reflexes should be considered as normal reflexes "deformed," in that the impulse has to follow another path owing to destruction of the usual one. If the ordinary cortico-spinal motor path be injured, the voluntary innervation and the reflexes of the extensor groups in the lower limb (Babinski's reflex, Strümpell's tibialis phenomenon, and Remak's femoral reflex) come under the control of a supplementary extra-pyramidal motor path. S. A. K. WILSON.

**APHASIA AND THE CEREBRAL ZONE OF SPEECH.** CHARLES (438) K. MILLS (Philadelphia), *Am. Journ. of the Med. Sci.*, Sept. 1904.

UNDER the above heading, a short article of the greatest interest is published by Dr Mills. It briefly records the clinical signs and the post-mortem changes found in a few cases of aphasia; in the rest of the article, the author gives a short summary of his deductions on the localisation of function in the (left) cerebral cortex.

This publication is of particular interest just now, in that it confirms by clinico-pathological evidence many of the deductions made (chiefly from the histological standpoint) by Dr A. W. Campbell, and communicated in an address by him delivered before the Neurological Society of the United Kingdom on March 17, 1904.

Both Mills and Campbell conclude that the speech centre is not restricted to Broca's convolution, but spreads forwards over a

considerable portion of the base of the third frontal gyrus and downwards on to the anterior part of the insula. Mills tentatively suggests that the hinder portion of the insula stands in the same relationship to the vestibular nerve as does the preinsula to the auditory; Campbell, on the other hand, has shown that histologically the hinder portion has temporal characters, and he is of opinion that it is audito-psychic, and that the central portion, having an entirely different structure, must have an entirely different function (he suggests that it may be gustatory).

The author brings forward much evidence to show that the graphic centre lies immediately in front of the motor centres for the hand and arm, and is thus in agreement with Campbell, who considers the whole strip of "intermediate pre-central" cortex, including Broca's area, to be cortex governing "the execution of skilled movements."

With regard to the localisation of auditory and visual functions in the cortex, Mills and Campbell are in complete agreement in regarding the cortex as containing both primary and secondary areas. For instance, the centre for primary recognition (hearing) is probably located in the caudal portion of the first temporal gyrus; and "the word-hearing centre is on the outskirts of this region at about the junction of the first and second temporal convolutions."

The author also maps out the psycho-visual area, giving somewhat tentatively definite localisation for word-seeing, letter-seeing, object-seeing, and naming of objects. The article is illustrated by several diagrams.

STANLEY BARNES.

#### **A CASE OF WORD-BLINDNESS, WITH RIGHT HOMONYMOUS**

(439) **HEMIANOPSIA.** By JAMES HINSHELWOOD, ALEXANDER MACPHAIL, and ALEXANDER R. FERGUSON, *Brit. Med. Journ.*, Nov. 12, 1904, p. 1304.

THE patient was a man of 58, a teacher of French and German. One morning he was startled to find that he could not read the exercise which a pupil had given him to correct. He took up a printed book and found he could not read a single word. When examined, he could not read even the largest letters of the test types, but could read at once the number standing at the top of each paragraph of the test types. He could also write correctly to dictation, but could not afterwards read what he had written. No other mental defect could be discovered.

There was right lateral homonymous hemianopsia, but no other defect of vision. The fundus appeared perfectly normal.

This was the condition in 1894, and he was under observation

until his death in 1903. No new symptoms arose during this time. He was unable to reacquire the visual memory of words, or at the most only of a very few of the short familiar ones.

His death occurred suddenly. He began to complain one evening of headache and giddiness, but nevertheless went out to visit a friend. At his friend's house he felt unwell, lay down, was carried home in a comatose state, and died in a few hours.

*Post-mortem Examination.*—The immediate cause of death was an extensive hæmorrhage from some of the superficial branches of the cerebellar arteries by which the greater part of the right cerebellar lobe was destroyed. The lesion round which the chief interest of the case centres was revealed on the inferior aspect of the left occipital lobe as a triangular area of sunken and atrophied appearance, over which the pia mater appeared to form the external wall of a cyst which completely masked the outline of the subjacent convolutions, and from which on puncture some three drachms of clear fluid escaped. This cyst was continuous with the posterior horn of the lateral ventricle, and probably arose from an acute softening due either to embolism or to thrombosis. The cortex thus destroyed may be summed up as a large triangular area limited by the calcarine fissure internally, and by the third temporal gyrus externally with the apex at the occipital pole; the cortex of the cuneus (above the posterior calcarine fissure and between it and the parieto-occipital fissure) appeared quite normal, though the fibres in the roof of the posterior horn of the lateral ventricle passing to and from the cuneus must have been encroached upon by the great dilatation of that cavity, while this same cause must have interfered, to a great extent, with all the fibres connected to what remained of the occipital cortex (including the tract usually called the optic radiation) and also the fibres ascending to the left angular gyrus from the left and right primary visual centres at the calcarine fissures.

*Remarks.*—The cuneus was quite intact except at the calcarine fissure. The lesion involved the calcarine fissure, although the greater part of it was infra-calcarine. This confirms to a certain extent the opinion of Henschen that the visual centre is localised in the cortex of the calcarine fissure and its immediate neighbourhood. The pathological examination also confirms the views of Déjerine and Sérieux with regard to two classes of word-blindness, viz., one in which the patient is word-blind and agraphic, and one in which he can still write to dictation, as in the present case. The left angular gyrus is now accepted as the area of visual memory for words and letters. When the angular gyrus is destroyed, the patient—according to Déjerine and Sérieux—is both word-blind and agraphic. They contend that before an individual can write he must be able to revive the visual memories of the words and

letters. Destruction of the cortex of the angular gyrus will render it impossible to revive these images, and hence writing is impossible. In the second class of cases the same writers maintain that the angular gyrus will be found intact, but that a lesion will be found beneath the cortex in such a position as to divide the fibres which pass to the left angular gyrus from the calcarine fissure in both occipital lobes, that such a subcortical lesion must almost necessarily involve the radiation of Gratiolet on the left side, and hence the patient has at the same time right lateral homonymous hemianopsia. The present case is a very beautiful demonstration of the correctness of their views. W. B. DRUMMOND.

**A CASE OF CONGENITAL WORD-BLINDNESS.** By JAMES HIN-  
(440) SHELWOOD, *Brit. Med. Journ.*, Nov. 12, 1904, p. 1303.

THE patient was a boy of 12 who had such difficulty in learning to read that, after seven years at school, he was only in Standard II., whereas he should have been in Standard V. In all other respects he was a quick and intelligent boy. He could rarely read by sight more than two or three consecutive words, but came to a standstill every second or third word, and was unable to proceed unless allowed to spell the word aloud, thus appealing to his auditory memory, or to spell it silently with his lips, thus appealing to his memory of speech movements. He spelt very well, and when asked to spell the words which he had failed to recognise by sight he nearly always did so without any difficulty.

It was advised that the boy, instead of being taught in class, should receive frequent short lessons by himself both at school, and again on his return home. Under this line of treatment he made good progress, and after two years' persistent training, was able to be raised to Standard IV. W. B. DRUMMOND.

**INEQUALITY OF THE PUPILS IN DISEASES OF THE LUNGS**  
(441) **AND PLEURA.** F. DEHÉRAIN, *Presse méd.*, Oct. 1, 1904,  
p. 630.

THE author found this symptom frequently in pneumonia. He observed that the mydriasis was not invariably on the side corresponding to the affected lung. He found pupil irregularity in three cases of acute bronchitis, lasting only till the infectious phenomena disappeared. The symptom was still more frequent in emphysema complicated with chronic bronchitis. It varied very much, but chiefly occurred during exacerbations. The patient sometimes complained of diplopia, muscæ volitantes, and other slight

disturbances of vision. In 26 cases of pulmonary tuberculosis out of a total of 120, inequality was observed. It is not necessarily an indication of apical disease. The persistence of the symptom in this affection was remarkable, but gave rise to no disturbance of vision. Lastly, in four cases of pleurisy with effusion it was present and lasted for several weeks. The explanation of the symptom probably is, that the sympathetic is excited in a mechanical way by the presence of enlarged glands in the case of phthisis and tuberculous pleurisy, or by other means in acute bronchitis and emphysema.

J. EASON.

**A CONTRIBUTION REGARDING CERVICAL RIBS.** HUGO LEVI, (442) *Neurol. Centralbl.*, Nov. 1904, p. 988.

THE case described is one of multiple sclerosis, in which there was a cervical rib. The rib on the left side formed a distinct tumour that on the right side was only detected by an X-ray examination. There was slight wasting of the muscles of the left thumb and little finger and a feeling of numbness at times without any objective sensory disturbance, however, along the wrist surface of the left forearm. Levi holds that the existence of a cervical rib in this case of multiple sclerosis is in accordance with the congenital theory of the pathogeny of the disease. A word of caution as to the interpretation of Radiograms is necessary. As the author remarks, the transverse process of the 7th cervical vertebræ stands out very prominently in an X-ray photograph and may be mistaken for an accessory rib, hence it is advisable to compare the photograph carefully with controls.

EDWIN BRAMWELL.

**FÆCAL VOMITING AND REVERSED PERISTALSIS IN FUNCTIONAL NERVOUS (CEREBRAL) DISEASE: A SUMMARY OF CASES AND CONCLUSIONS.** F. PARKES WEBER, *Brain*, Part cvi., 1904, p. 170.

THE writer gives a resumé of the recorded cases bearing upon this matter, in which formed fæces, enemata, suppositories and other substances were passed upwards from the rectum and expelled by the mouth. These cases number about twenty-seven, and the writer records with special minuteness one observed by himself, which is typical of the others. The patient, a young woman, aged 22, of a markedly hysterical temperament, repeatedly vomited fæcal masses, and ten minutes after an enema containing methylene blue, the vomit was tinged of this colour. Owing to the aggravated abdominal symptoms no less than three laparotomies



were performed on her, but at none of these was there found any abnormality or any fistula between the great intestine and stomach through which the faecal material could have passed. The case showed other hysterical manifestations, and was ultimately much benefited by isolation treatment. In other cases evidence of a spasmodic stricture in the bowel has been afforded by operation or otherwise.

The writer maintains that these exaggerated cases of faecal vomiting are associated with functional nervous conditions, organic obstruction being accompanied only by an admixture of faeces with the vomit, which contains no definite faeculent pieces. He adduces a mass of evidence to show that the ileo-caecal valve in these cases is incompetent either congenitally or as the result of distension of the colon or of muscular contraction at the valve, and that the passage of faeces, etc., upwards is due to anti-peristalsis.

He concludes that faecal vomiting of this extreme type is simply an exaggerated type of hysterical vomiting.

JOHN D. COMRIE.

**ATROPHY OF THE MUSCLES OF THE THIGH AND RIGHT  
(444) LEG FOLLOWING A FALL—CONTRACTURE OF THE  
FOOT—TREATMENT BY SINUSOIDAL VOLTAISATION.**

Dr THIELLÉ, *Bulletin Officiel de la Société Française d'Électrothérapie et de Radiologie*, July and August 1904.

THE patient, a spinster, æt. 35, enjoyed good health till February 10, 1899, when, while descending the stairs, she fell with her leg twisted under her. Her doctor diagnosed a sprain, and the usual methods of treatment were employed.

On June 10, she was seen by Dr Thiellé. At this date she was still unable to walk alone, and could not rest her weight on the foot. She complained of pain, specially at night, behind the external malleolus and in the big toe, but the joint movements were painless, and neither the sciatic nor its branches were painful on pressure. There was reflex contracture of the foot, and the thigh and leg were wasted. The X-rays showed no lesion of the external malleolus.

*Treatment.*—From 16th to 29th July 24 hydro-electric baths were given, the first 6 being with the oscillating current in order to soothe the pain, and the latter 18 with the alternating sinusoidal current. After the fourth bath the pain completely disappeared, and on the 25th July the thigh and calf of the affected leg were almost equal in size to those of the other side. In May 1900 the two legs were of equal size, and she could walk easily.

A. DINGWALL FORDYCE.

**SOME REMARKS ON THE THEORY OF MDLLE. JOTEYKO  
(445) REGARDING THE MECHANISM OF THE REACTION  
OF DEGENERATION. MM. LAQUERRIÈRE and DELHERM,  
*Bull. Offic. de la Soc. Franç. d'Électrothérapie et de Radiologie*,  
July 1904.**

In her paper published in the *Bulletin de l'Académie royale de Médecine de Belgique*, 26th December 1903, Dr Joteyko arrives at the conclusion that "the reaction of degeneration is present coincidently with an histological condition in which the degenerated muscle comes appreciably to resemble non-striped muscle, and, like it, gives a reaction due to stimulation of the sarcoplasm."

Putting aside at once the view that the "abnormal" reaction of a muscle whose nerve supply has been cut off may be regarded as the "normal" reaction of a muscle apart from its nerve, she proceeds to state her view that there exists in muscle itself, apart from nervous structures, two elements which react differently to stimulation. She considers that the anisotropic fibrillary substance and the sarcoplasm both contract under stimulation, but contract in different manners. Non-striped muscles, composed largely of sarcoplasm, give scarcely any response to isolated induced shocks, but react to the faradic current when of considerable frequency; they react to the constant current (if the period of stimulation is not too short), and they react more to the positive than the negative pole, at the same time exhibiting a gradual and sluggish contraction; the striped fibre reacts readily to isolated induced shocks, and obeys the laws of polarity of Pflüger and Chauveau, and exhibits a sharp contraction.

Thus the characteristic reactions of degenerated striped muscle approach in type the phenomena observed in the case of non-striped muscle, and, as recent histological researches have shown, when a muscle degenerates after section of its nerve, there occurs diminution or disappearance of the fibrillary substance and great development of sarcoplasm. With these views the authors of this paper are in full accord, but wish to bring forward one objection.

They maintain that without further information it is impossible to conclude that the electric reactions are absolutely similar in the case of degenerated muscle and non-striped muscle, because, while the non-striped muscle gives a reaction during the period of flow of the constant current, the sarcoplasm of the degenerated muscle shows no such reaction, and only reacts at the periods of opening and closing the current or during rapid variations in its intensity.

A. DINGWALL FORDYCE

**THE RELATION OF ALCOHOL TO ARTERIO-SCLEROSIS.**

(446) RICHARD CABOT, *Journal of Amer. Med. Assoc.*, September 17, 1904, p. 772.

MOST text-books assert the importance of alcoholism in the etiology of arterio-sclerosis. To test the accuracy of this view the writer studied first 283 cases of severe chronic alcoholism at Bridgewater State Farm, and the Foxboro Asylum for dipsomaniacs. Cases over fifty years of age and those who had a history of syphilis being excluded, it was found that only 6 per cent. showed arterio-sclerosis; while if the cases under forty years of age were taken, the percentage was as low as 1.4. In the second place, to determine in what proportion of cases of relatively early arterio-sclerosis of peripheral arteries alcoholism was present, forty-five cases of arterio-sclerosis examined at the Massachusetts General Hospital gave only a percentage of thirteen patients with any history of alcoholism. The records of the 656 cases at clinico-pathological laboratory of Massachusetts General Hospital were next examined. It was found that of these only ninety-five or 14.5 per cent. were under fifty years of age, and that out of these ninety-five cases in which arterio-sclerosis was found at autopsy, only twenty-one per cent. had a marked alcoholic history. This percentage was further reduced to seventeen if cases with chronic nephritis were excluded.

In the discussion which followed the paper, Dr Alfred Stengel and others maintained the view that alcohol is a factor in the production of arterio-sclerosis.

A. HILL BUCHAN.

**PSYCHIATRY.****THE PROBLEM OF HEREDITY FROM THE PSYCHIATRICAL**

(447) ASPECT. W. KÖNIG, *Brit. Med. Journ.*, Oct. 15, 1904, p. 965.

THE author in this paper examines the bearing of Dr Beard's findings on insane heredity viewed from the clinical aspect, and considers the question of mental heredity with special reference to the inheritance of acquired qualities.

By proving the direct continuity of germ cells from generation to generation, and establishing antithetic alteration of generations in the metazoa, Dr Beard, says the author, has displaced theory by solid anatomical facts, and given us a clearer insight into the problem of heredity.

The author distinguishes three principal groups of heredity—homologous, dissimilar, and what he terms "mixed" heredity, that is, homologous heredity on the side of one parent, and dissimilar on the part of the other. He confines his attention chiefly to the first

group—ancestor and descendant exhibiting the same variety of mental affection. He discusses two main questions: (1) Is there any clinical evidence of acquired mental abnormalities being transmitted to the offspring? (2) To what extent in insanity does environment influence the germ cells, and under what circumstances does it affect the same?

The author's experience is based on the histories of 3329 cases. As regards the etiology of adult general paralysis—a true type of acquired mental derangement—the author is in complete concurrence with those who hold that in most instances the syphilitic virus is a necessary step in the production of the disease. Environmental surroundings, such as stress and inebriety, he regards as predisposing factors.

Among 1151 male and female paralytics, some had congenitally weak minds, and sometimes there was a history of convulsions. In very numerous examples a distinct family history of insanity has been obtained. The author finds that the offspring of paralytic parents are frequently imbeciles, idiots, sufferers from early epilepsy, chorea, and various other neurotic ailments. Among the adult descendants he has noted a fair number of paranoiacs; but on the other hand, a not infrequent record of sound children could be obtained, and he quotes one instance of paralysis in a parent who had seven healthy children, and no miscarriages.

The author then gives a string of genealogic examples in which the paralytic descendants exhibit various forms of heredity, after which he gives some cases of homologous heredity; and he considers it worthy of note that quite a number of these homologous cases, both ascendant and descendant, had a clear history of syphilis, all these examples forcibly pointing to an inherited disposition, of which in some families all descendants participate, and which increases the chance of syphilitically infected descendants to develop insanity. The author thinks that these are conclusive reasons for assuming that, in all individuals burdened with heredity, the parental germ cells may be adversely affected, and so encourage parasymphilitic changes in the infected offspring, this being particularly true in instances of homologous heredity. Speaking next of alcoholic indulgence, and having inquired into the history of 536 cases whose insanity was attributed to steady abuse of intoxicants, the main facts elicited were, an enormous prevalence of homologous, namely alcoholic heredity, a rather low rate of hereditarily untainted individuals, and an intermediate proportion of dissimilar heredity. Among the descendants he has found instances of true paranoia, katatonia, dementia præcox, epilepsy, and quite a fair sprinkling of organic cerebral disorders, including general paralysis. Speaking of the association between alcoholism and epilepsy, he finds that in 111 instances, fits were developed in the course of

an intemperate life; the evidence of clinical experience disclosing the fact that the majority of habitual drinkers have a neurotic history, and that the severity of the heritage is more prominent in those having a direct alcoholic ancestry. Moreover, says the author, clinical study does not support the suggestion that the acquired habit of drink, or even any craving, is transmitted, but the inference is rather in favour of the germ cells being so modified as to render the offspring particularly liable to the injurious influence of alcohol. Alcoholism, he says, is largely the product of environmental agency, based on a neurotic diathesis. No other form of mental irregularity is to the same extent dependent on the environment. He then speaks of the disastrous consequences of heredity in idiopathic epilepsy, and gives characteristic examples of cumulative heredity in epileptics, sometimes with an alternation of homologous and dissimilar heredity in grandparent and parent.

In the author's opinion ancestral tuberculosis is of some hereditary importance generally, and distinctly so in the history of idiocy and infantile cerebral paralysis, in assisting the formation of neuropathic disposition. In cases of acquired epilepsy, there is a large proportion with a neuropathic history; but among those with a "clean bill" of heredity, he has not met with a single instance where the acquired disease was manifestly handed down to the offspring. He assumes, therefore, that in idiopathic epilepsy the germ cells are so seriously modified, that the inherited disposition is duly developed. With regard to post-traumatic neuroses there is no proof of hereditary transmission. Respecting the group of senile dementia and other forms of insanity due to arteriosclerotic changes, frequently there is found a hereditary predisposition for atheromatous degeneration.

Paranoiacs, and most sufferers from the types of functional disturbance, show a more or less powerful hereditary disposition to insanity and other nervous disorders. Sometimes—take a typical case of essential paranoia—the morbid character of the germ cell will probably develop at one time or other, in the face of the most favourable surrounding factors; while in other cases, *e.g.* in the litigious type of paranoia, environmental agents may give instigation to a gradually progressive development either of a dormant disposition or of a rudimentary disorder hitherto less conspicuous, and perhaps of a stationary character. In summing up, clinical experience, says the author, does not clash with the results of Dr Beard's investigations. First, there is no clinical evidence of acquired mental abnormalities being transmitted to the offspring. Secondly, it is highly probable that "the influences of the environment are reflected on the germ cells." Thirdly, the hereditary potentialities of the germ cells may in some cases develop in early or later life, unaided by any traceable environmental influence—

in other cases they certainly remain dormant, or in a rudimentary state of development, until roused to life by inimical extrinsic factors.

G. RUTHERFORD JEFFREY.

**ON THE PSYCHOSES RESULTING FROM SYPHILIS.** By G. (448) ANGIOLELLA and R. GALDI, *Annali di Neurologia*, 1904, f. 3.

THE authors, after reviewing the literature dealing with the occurrence of insanity in the second stage of syphilis, describe eight cases of this kind which have come under their observation. All of the cases recovered under anti-syphilitic treatment. They also describe five other cases of insanity in syphilitic subjects, in which the clinical and pathological features seemed to be intermediate between the acute psychoses represented in the first group and general paralysis of the insane. These cases were only temporarily benefited by anti-syphilitic treatment, and one of them, at least, ultimately became a general paralytic. Lastly, they describe a case of hypochondriacal delirium resulting from syphilis, which is cited to show that this infection is capable of producing a purely psychical influence upon the individual.

The authors conclude that syphilitic infection, acting simply as a cause of toxæmia, is capable of producing certain forms of insanity, which clinically and pathologically resemble other toxic insanities. These psychoses resulting from syphilis are varied in their symptomatology, and therefore do not form a definite clinical group; they can only be classed together on the ground of their etiology. They manifest themselves as states of excitement, depression or confusion; there may be a single attack, or the disorder may assume a recurrent form. In other instances the clinical picture is one which closely simulates that of general paralysis, both as regards dementia and physical symptoms. They think that this morbid condition gradually passes into general paralysis, and that its occurrence throws light upon the syphilitic pathogenesis of the latter.

W. FORD ROBERTSON.

**CLINICAL AND PATHOLOGICAL CONTRIBUTION TO THE (449) STUDY OF THE RELATIONS BETWEEN SYPHILIS AND PROGRESSIVE PARALYSIS.** By RODOLFO STANZIALE, *Annali di Neurologia*, 1904, f. iv.

IN 100 cases of general paralysis studied clinically by the author there were 70 in which the occurrence of syphilis was certain, and 17 others in which it was probable. In 32 cases, syphilis seemed the only etiological factor, whilst in 38 it was associated with other factors, such as neuropathic heredity and alcoholism. He

was unable to observe any modification in the course of the disease from the therapeutic use of mercurials in large doses.

The cerebral arteries were examined histologically in nine cases. In six of these, well-marked morbid changes were found. There was a condition of periarteritis which in its initial phases, and especially in the arterioles, manifested itself as a round-cell infiltration, and in its later phases as a simple fibrous thickening. The media was often normal, but when the changes in the other coats were advanced it tended to be involved in the morbid process, becoming thinner and sometimes disappearing. The internal elastic lamina was commonly not only thickened, but showed the formation of several new layers. The intima was the seat of hyperplastic thickening, consisting at first of round-cells and in the later stages of fibrous tissue, either localised or extending along a considerable tract of the vessel. Miliary aneurisms were frequently observed in the medium-sized and small arterioles. In the other three cases the arterial changes were very slight, consisting of some local fibrous thickening of the intima or adventitia.

The author discusses the question of whether from the histological characters it can be deduced that an arteritis is of syphilitic origin, and favours the view that, whilst no single feature is diagnostic, the existence of a certain complex of arterial changes warrants the conclusion that the morbid condition is of this nature. This complex of arterial changes is essentially that which was found in the cerebral arteries of six cases out of nine examined histologically. As, however, similar arterial changes may be found in cases of cerebral syphilis which do not present either the clinical picture of general paralysis or the special histological changes that affect the nervous parenchyma in this disease, he infers that there must be other hereditary or acquired factors, in consequence of the action of which the vascular lesions are accompanied by the special parenchymatous changes. At the same time, since it appears that syphilis may be the sole etiological factor, it cannot be excluded that in some cases this infection influences the nervous system so disastrously that recovery from the degenerative process which is initiated becomes impossible.

W. FORD ROBERTSON.

**CRIMINAL RESPONSIBILITY.** Dr MERCIER and others, *British* (450) *Medical Journal*, 15th October 1904.

At the Oxford Meeting of the British Medical Association, Dr Mercier, President of the Section of Psychological Medicine, opened a discussion on this subject by an important and singularly able paper. He first indicated the different points of view from which the medical and the legal mind regard this question, the medical

mind regarding primarily and perhaps too exclusively the individual offender, whilst to the legal mind the protection of the community from wrongdoers is the main end. These are, however, Dr Mercier stated, merely two aspects of the same problem, and neither of them the highest or ultimate aim of the treatment of crime, which should always have for its principal objective the strengthening of the sense of responsibility throughout the community. He then mentioned some factors which tend to the disintegration of this sense of responsibility, and in an amusing phillipic, Carlylean in intensity, and—if one may venture to say so—unconscious exaggeration, inveighed against the modern doctrine of degeneracy—stigmatic degeneracy—in particular as applied in the Elmira reformatory, and in picturesque and forcible language deprecated the burdening of honest tax-paying citizens with the provision of Turkish baths, musical drills, and other luxuries of “criminal pampering” for the questionable benefit of converting a few criminals into indifferent citizens. This doctrine with its—for Dr Mercier—inevitable result of weakening the sense of responsibility he stigmatised as most pernicious and immoral. The elements of punishment he left in other hands, and concerned himself with the questions, “Who ought to be punished?” and “With how much punishment?” To the first question he replied, “Wrongdoers, by which is meant those who for their own gratification and without justifying provocation wilfully do harm to others.” This definition, he said, excludes from wrongness certain cases of doing harm, and provides the same criterion of responsibility, that is of liability to punishment, to both the sane and the insane. “The responsibility for guilt lies in the *animus nocendi* or *scelerandi*.” He then interpreted in this light, and defended, the much disputed answers of the judges in 1843 to the question, “What are the proper questions to be submitted to the jury when a person afflicted with insane delusion respecting one or more subjects or persons is charged with the commission of crime, and insanity is set up as a defence?” which were to the effect that it must be clearly proved that the party was labouring under such a defect of reason as not to know the nature and quality of the act he was doing. Dr Mercier maintained the adequacy of this pronouncement, and on this basis formulated the following: Firstly, harm done, with no intention of obtaining gratification by the doing, is not wrong and is not punishable. Secondly, provocation or threat justifies retaliation in measure—in such measure as will prevent the threatened injury—and the exoneration is complete whether the belief be true, a sane mistake, or an insane delusion. Thirdly, the harm must be done wilfully, or there is no wrong done; wilful action implying knowledge of the nature of the act and its natural consequences, and also volition. This third dictum, which



covers obviously the bulk of the debatable matter, was discussed at considerable length, and the author showed, with regard to knowledge, that in certain cases the act may be recognised by the actor as illegal, yet may not be appreciated in its true proportions; that is, that there is present a mental confusion, "and it is this inability to think clearly about anything connected with the delusion which prevents the insane malefactor from knowing how wrong is his act." These persons were not as such, in his opinion, necessarily exempt from punishment, yet they ought to be partially exonerated from any misdeed they may commit.

With respect to will, Dr Mercier emphasised the fact that criminal law takes account of those acts only that are wilful, and in saying so desired that he might not be understood as advocating the doctrine of irresistible impulse as the only alternative defence to the ignorance of right and wrong. Irresistible impulse he regarded as so rare—except in cases of unmistakable insanity—that the question of irresponsibility scarcely ever arises in a court of law; and that in the majority of cases where this has been pleaded, absence of will or *animus nocendi* was the condition actually present. Of irresistible impulse in the technical sense of a sudden violent outburst he was unable to cite a single example, but of the existence of obsession, irresistible but not necessarily impulsive, there is, he said, not a shadow of a doubt. Lastly, he discussed briefly cases of morbid desire. "Compromise," he said, "does not usually commend itself to me, but in this case I do think that safety lies in the middle way. Experience shows that excessive severity is not an effectual deterrent; and most of us would recoil from treating with great severity acts which we feel to be, in part at least, the outcome of disease. Nevertheless, universal experience forbids us to suppose that desire cannot be resisted by will, or that the will to abstain from action may not be reinforced by fear of incurring imprisonment."

Sir Herbert Stephen continued the discussion, and said he found himself in almost complete agreement with Dr Mercier's views, and remarked, firstly, that if there were a "criminal neurosis"—and following the opinions of Dr Maudsley and Dr Mercier he did not believe it existed—he should advise juries that this was evidence tending to prove the accused to be guilty, and would also regard its existence as a reason for his prolonged detention; and secondly, that some criminals who were insane should be treated as criminals, though it might happen that their insanity should be considered as a mitigating circumstance. He then instanced three typical classes: (a) of epileptic origin in which the crime was committed during temporary unconsciousness, and in which case he advocated medical treatment solely, with such precautionary restraint as might be necessary; (b) transitory obsession, such as

child-murder following child-birth, in which case he recommended a period of seclusion and discipline; and (c) cases where the insanity was a much more remote cause of the crime, and where either some or no provocation had been received by the malefactor. These he considered ought to be punished, in some cases with full penalty, in others with mitigated force. He would treat them, not as invalids unfortunately concerned in crime, but as criminals who happened to be suffering from a disease which might or might not be a reason for mitigating the severity of the punishment. These opinions of Dr Mercier and Sir Herbert Stephen evoked the most direct opposition. Mr Douglas Cowburn considered that insanity necessarily exonerated from punishment, and said that the answers of the judges in 1843 had been repudiated by many of the States of America, by Italy, and even by Russia, and submitted that the only real test was the law laid down by the State of New York, that "no act done by a person in a state of insanity could be punished as an offence." This left the decision as to the sanity or insanity of the accused entirely in medical hands and the question of responsibility and punishment in those of the law. Mr Havelock Ellis, who identified himself with the views of Aschaffenburg, was strongly of opinion that the only agreement possible between legal and medical opinion was an agreement to retain quite different points of view, that the medical man was concerned solely with facts, and that the determination of responsibility—which was not a medical but a metaphysico-legal conception—rested with the law, acting in accordance with the facts supplied by medical opinion. The discussion was continued by Doctors Savage, Claye Shaw, Yellowlees, Conolly Norman, Blandford, Jones, Shuttleworth and Prof. White, all of whom dissented in varying degrees from the views of the President.

The criticism turned mainly on three points: firstly, the existence of a class of degenerates—and those who have an intimate acquaintance with prison populations know how considerable this class is—who are not, in the present state of lunacy law, certifiable; who are by innate character at perpetual variance with the laws which govern and have been formulated by the majority; who are the subjects of what Sir Herbert Stephen alluded to as a "criminal neurosis," and who are only slightly, often not at all, corrigible: secondly, the essential solidarity of the mind and the consequent illogicality of holding an insane person responsible for his actions; and thirdly, the allocation of questions of responsibility, the alteration in the psychological constituents of responsibility being quite as much a question for the consideration of the medical man as the lawyer.

With regard to Dr Mercier's anathema of the tenet that there are people, not certifiably insane, who are abnormal in mental

make-up, who very often exhibit a collection of the so-called stigmata of degeneration he so amusingly derides, for whom punishment in the ordinary sense is utterly futile and who cannot be considered responsible, it is a little difficult to see why a recognition of this fact, or no-fact, as Dr Mercier would have it, should have the disastrous effects Dr Mercier apprehends. Surely, as Sir Herbert Stephen has said, it should result in their "being deprived of their liberty to commit crimes for as long a period as seems convenient, subject to their treatment being as little injurious to them as is consistent with public policy," and the "weakening of the sense of responsibility" can hardly affect a malefactor in whom, *ex hypothesi*, it already is practically *nil*.

R. CUNYNGHAM BROWN.

### TREATMENT.

**FURTHER NOTES ON THE TREATMENT OF BIRTH PARALYSIS (451) OF THE UPPER EXTREMITY BY SUTURE OF THE FIFTH AND SIXTH CERVICAL NERVES.** ROBERT KENNEDY, *Brit. Med. Journ.*, Oct. 22, 1904, p. 1065.

THE treatment of Duchenne's birth paralysis of the upper extremity by suture of the brachial plexus was first published by the author in a paper which appeared in the *British Medical Journal* in January 1903. In the present paper the further histories of the three cases there published are given, together with the histories of two new cases.

Of the five cases recorded, all progressed or are progressing satisfactorily, except one in which the result was interfered with by compression of the seat of suture by cicatrisation external to the nerves. This case was re-operated upon, but at a date too recent to allow of the ultimate result being recorded in this paper.

As regards the exact seat of the lesion, the author confirms the view that it is situated at the junction of the anterior primary divisions of the fifth and sixth cervical nerves, this portion having been found to be affected in all the cases upon which he has operated.

The operation recommended by the author is excision of the scar on the nerve trunks and suture of the divided ends. He points out that the method of nerve anastomosis is not to be recommended in such cases.

As regards the most suitable age for operation, the author recommends that the operation be not performed till two months of age, in order to allow of the possibility of signs of spontaneous recovery being exhibited. The interval of time elapsing between the operation and first sign of restoration of the muscles increases

in a certain proportion, according to the interval which has elapsed between the accident and date of operation, up to a certain limit. Therefore the earlier the operation, the shorter will be the interval which will require to be passed after the operation before the first signs of recovery are exhibited.

AUTHOR'S ABSTRACT.

**THE FUNDAMENTAL PRINCIPLES OF ELECTROTHERAPY IN  
(452) CASES OF NERVOUS DISEASE.** M. E. DOUMER, *Journ. de Neurol.*, October 5, 1904, p. 361.

THE first half of his paper M. Doumer devotes to combating the idea that the beneficial effects of electric treatment are due to psychical influence—suggestion.

He lives in the hope of seeing electrotherapy give birth to electrophysiology, and in his present paper seeks to prove "in a spirit of modesty" that electricity in itself possesses curative properties.

In addition to the chemical actions associated with the phenomena of electrolysis, undoubtedly the beneficial effects of electric action are in no small degree attributable to the direct action of electric energy itself.

Unfortunately, however, with such a strong case behind him, M. Doumer, while striving after modesty, succeeds rather in impressing the reader with the idea that his arguments are lacking in weight.

In the second part of the paper he lays great stress on the fact, which has hitherto received less than its due amount of recognition, that the electric current is not a remedy to be used solely in affections of the nervous and muscular systems.

Many facts can be adduced to prove that electricity has a direct action on living cells, and that it acts on all the cells of the body, and not in any exceptional manner on those of the nervous and muscular systems.

Naturally, the effects on these systems are more readily observable than the other effects, but none the less are the general beneficial effects present throughout the organism.

M. Doumer gives, as the two fundamental principles on which is based general electrotherapy:—

(1) That in acute disease, or where inflammation is present, cure or improvement is more rapid than where these signs are less marked.

(2) That in the treatment of an inflammatory condition the first result obtained is always disappearance or diminution of the inflammation.

Applying these principles to the treatment of nervous diseases,

he recommends in all cases, such as infantile paralysis, cerebral hæmorrhage, etc., that, immediately following the onset of the illness, energetic electric treatment should be adopted.

By such action he claims to have obtained much better results than are possible by following the established custom of deferring electric treatment till the acute stage of the illness is over.

The word of the French savant must of necessity carry considerable weight with it, alike by virtue of his great experience and eminent ability, but, nevertheless, the day seems still distant when we shall see his somewhat heroic recommendations carried out in routine practice.

A. DINGWALL-FORDYCE.

**THE ELECTRIC TREATMENT OF FACIAL NEURALGIA—AN  
(453) IMPROVED METHOD. A. ZIMMERN, *Archiv. d'Électricité Méd.*,  
Sept. 25, 1904, p. 698.**

In clinical practice the physician meets with two forms of facial neuralgia—(1) the slight and (2) the severe form.

The former may be very persistent and last even for years; it may occasion very considerable pain, and may simulate the severe form; and, further, it is of the same nature as certain forms of sciatica and very persistent forms of intercostal neuralgia.

The latter—severe form—is, on the contrary, homologous with no affections of other nervous districts. It is the epileptiform neuralgia of Trousseau, and is frequently accompanied by tic.

In the slight form, electric treatment, as a rule, ensures complete and final cure, with absolutely no future need of this treatment.

In the severe form, the beneficial effects of treatment by the continued current are evidenced by the diminished amount of pain and the smaller number of crises. In many of these cases, after careful regular treatment, the attacks are warded off for a certain period; but, as a rule, they recur, though usually in a much milder form, and by treating such cases every two months for ten days or so at a time the attacks are still farther reduced, both in number and severity. Professor Bergonié has described this treatment as palliative—a term which is strictly accurate, but perhaps somewhat unduly modest.

As regards technique—the large facial plate of Bergonié, which is half-mask in shape and can be moulded on the face, is the method of application which has been used.

Each application was of an hour's duration, the strength of the current employed varying from 5 to 10 m.a., according to the case.

In most cases of the severe form of facial neuralgia it was

difficult to obtain rapid improvement, and, as a rule, treatment had to be continued for at least a month, and usually two or three months, before benefit was marked and the patient sensibly relieved.

In cases of the slight form, at least eight to ten applications of the continued current were necessary to conclude the cure.

The following treatment has in many cases given more rapid results than that by the above method.

It consists of hydro-electric injections into the nostril of the affected side, or into the space between the gums and cheek.

A vessel filled with salt solution (7 in 1000) is connected by an india-rubber tube with a glass cannula—similar to that used for inhalations, when nasal electrification is to be undertaken. For intra-buccal applications the cannula is longer, flattened, and so shaped at the end that a thin sheet of liquid is obtained. From the orifice of the cannula a thread of silver wire passes up the rubber tube and is connected with the positive electrode; the negative electrode is placed on the nape of the neck.

The patient holds the cannula in his hand, and, with the stop-cock on the tube half open, sprays the mucous membrane.

The strength of current used varies from 5 to 15 m.a., and the application lasts about an hour, the cannula never being kept quite motionless in order to prevent scarring.

As yet it is impossible to accurately estimate the value of this method of treatment, but the author is convinced that it causes rapid amelioration of the painful symptoms.

In cases of the severe form it is unwise as yet to speak definitely as regards the result of treatment, as occasionally in this form spontaneous improvement occurs which might erroneously be attributed to the electrification.

As regards cases of the slight form of facial neuralgia, however, the effects are indisputable. Electrification of the nasal or buccal mucous membrane by the hydro-electric method brings about much speedier cure than do the external applications, and after three or four applications persistent neuralgias of several months' duration can be completely and finally cured. This rapidity of action induces the author to recommend this procedure above all others for the treatment of the slight form of facial neuralgia.

A. DINGWALL-FORDYCE.

**SCIATICA TREATED BY THE OSCILLATING CURRENT.** Dr (454) THIELLÉ, *Bull. Offic. de la Soc. Franç. d'Électrothérapie et de Radiologie*, July and August 1904.

THE current was applied by means of a bath for 35 to 40 minutes at a time, the electrodes being placed beside the affected limb.

After 25 to 30 minutes' application the current was reversed and applied for 10 to 15 minutes. The maximum intensity employed was 120 m.a. After even the first application the patient experienced distinct relief of pain in the leg, which, however, only lasted for a few hours, but the pain on returning was of a less severe character than formerly.

As a rule the sensibility to the passage of the current was diminished in the affected leg. For a first application it was found advisable that the intensity of the current should not exceed 45 to 60 m.a.

The duration of treatment was very variable; in acute cases cure was rapid, while in old-standing cases it was very much slower, and in some cases not complete. The author considers that this current has an action similar to but less marked than the alternating sinusoidal current. He cites 13 cases of sciatica treated by this method.

A. DINGWALL FORDYCE.

**THE USE OF SPINAL ANÆSTHESIA IN LABOUR.** A. MARTIN,  
(455) *Münch. med. Wchnschr.*, Oct. 16, 1904, p. 1817.

THIS paper, in which Martin records his impressions of the value of spinal anæsthesia from its use in over thirty cases of labour, is indirectly of considerable neurological interest. A combination of adrenalin and cocaine was employed for the injection. 1 c.c. of a 0·5 in 1000 solution of adrenalin was first injected into the spinal canal, and, five minutes later, this was followed by an injection of cocaine in a dose varying from 0·8 of a gramme to 2 grammes. Among the thirty cases, of which half were primiparæ, all healthy women whose ages ranged from eighteen to thirty-six years, complete anæsthesia was obtained, extending commonly up to the lower ribs, and occasionally so widespread as to render the whole trunk analgesic. In one case only was the anæsthesia confined to the genital organs alone. The time of duration of the anæsthesia was sometimes not much more than half an hour, but at other times lasted as long as four hours. Martin finds that pregnant women bear spinal anæsthesia very well, and disagreeable after-effects are not usual. Occasionally very severe vomiting occurred, but rigors with rise of temperature did not appear in any of the cases.

As regards the effects of this form of anæsthesia on the course of labour, it was noted that the activity of the uterus was somewhat impaired, both in the number and force of the contractions. The "bearing down" pains were specially affected. If the labour was completed within one and a half hours after the injection, the

women had hardly an unpleasant sensation in the whole process of delivery, even when this entailed the use of forceps, version, etc. Spinal anaesthesia in no case led to the occurrence of anxious symptoms during the third stage of labour, and the processes of uterine involution and of lactation were in no way disturbed. Martin considers that this method of anaesthesia is as good as chloroform narcosis, and when still further tested it will become more apparent that it produces no harmful results. Now that he has had some experience of its use in physiological cases, he intends to test its value in cases of pathological labour.

OLIPHANT NICHOLSON.



## Review

**HEREDITY AND GENETIC VARIATION.** AUGUST WEISMANN,  
*Vortraege ueber Deszendenztheorie*, 2te verbesserte Auflage, mit  
3 farbigen Tafeln und 131 Textfiguren. Jena: Gustav Fischer,  
1904.

SCARCE two years have passed since Weismann gave to the world the text (in two volumes of some 900 pages) of his lectures on descent and heredity. In the opening words of the preface the author described it as his legacy to those who shall come afterwards. The handsome and wonderfully cheap volume before us contains in barely 700 pages his revised version of this gift of his researches and still more of his mental labours. The field traversed in the work is very wide, for, indeed, the lectures deal with far more than the theory of descent or Darwinism, as they originally did some twenty years ago, when as a wandering student the writer sat at Weismann's feet. They have become at the same time a theory, an impossible one, of the mode of the development of the higher animals, and a very complete, a highly improbable one, of heredity and genetic variation. In these latter things Weismann goes far beyond anything Darwin ever attempted. To Darwin the science of animal development was a sealed book, while for his sole contribution to a knowledge of heredity, the provisional hypothesis of pangenesis, it cannot be claimed that in it he did more than endeavour to form a conception of how the phenomena might come to pass. For a quarter of a century and more Weismann has been a leading, the chief, exponent of Darwinism. In his eyes the theory of natural selection was "all-sufficient." In these and similar words he defended it against the attacks of the late Herbert Spencer, and only now, in the preface of the second edition of his "Vortraege," do we meet with an admission that something more is required than "natural selection." He writes: "To-day there are more than a few who regard the principle of selection as an exploded idea, because they consider that the Darwinian 'natural selection' alone does not suffice for the explanation of organic development. In the latter, it appears to me, they are right, only they overlook that the principle of 'natural selection' is capable of a considerable extension and deepening, which cannot without

examination be brushed aside." In none of his earlier writings—not even in the first edition of the present work—will the reader find anything at all like this significant confession. In the popular mind the Darwinian theory is identical with that of the evolution of life, of which it really professes to be, and is, only one of many conceivable explanations. In the same way Weismann sees in his theories of the germplasm and of germinal selection, in those of heredity and development based on the biophores, ids, idants, etc., only another, perhaps slightly revised and extended, version of the Darwinian theory of natural selection. But there is an enormous difference between Darwin's theory of natural selection and the intricate and wonderful theories of Weismann. Within limits the premisses of Darwin seem sound enough, but the conclusions do not of necessity follow from them, while Weismann's conclusions would inevitably follow from his premisses were it admitted that the grounds upon which these rested were anything more than unproved, and in the majority of cases unprovable, hypotheses. Few disciples of Darwin—not even Weismann—follow out the principle of natural selection to its logical limits. This attempt has really only been made by Dr Archdall Reid, whose position is safe from attack, if the validity of his premisses be granted. The argument is practically, but not verbally, given in the following: "There are always the unfit and the fit. Under all conceivable circumstances the fit survive, while the unfit are eliminated. Therefore, alcohol, opium, malaria, toxins, etc., have no influences on the race, for they eliminate only the unfit, and the fit survive." To clinch the argument, there remains but to add the mathematician's "which is as it should be." This is Darwinism carried out to a logical conclusion, and with a vengeance! The second premiss is that upon which the reasoning collapses, whether carried to a logical extreme, as is done by Archdall Reid, or whether the argument be reinforced by "germinal selection" and other unseen forces, as advocated and believed by Weismann. Granted that both fit and unfit individuals obtain, there is no evidence to show (1) that the unfit are eliminated before they breed, or (2) that the fit survive no matter how adverse be the conditions of life. Applying this to man, to grasp its true significance one has but to read that intensely pathetic book of horrors, "The People of the Abyss."<sup>1</sup> The process of elimination, at times rapid, always remorseless, is often exceedingly slow, if sure: it seeks out its victims not only among the non-breeding and presumably unfit individuals, but it also fastens its merciless fangs in the fit, who are merely unfortunate.

Turning to the purely scientific aspects of Weismann's book, whilst it is, even to the professed zoologist, replete with interest,

<sup>1</sup> Jack London, "The People of the Abyss." London: Labister & Co. 1903.

one cannot study it without feelings of intense disappointment. The external world, "the conditions of natural existence" (Semper)—in a word, the influences of the environment—exist hardly more for Weismann than they did for Darwin and Wallace. Even such obvious adaptations to environment as those furnished by the facts of mimicry require "germinal selection" and its unprovable hypotheses to account for them. Weismann still lives and moves in the researches and advances, in many of the groundless dogmas and erroneous doctrines of the nineteenth century. With the exception of an occasional brief reference to some supposed recent or usually merely foreshadowed important discovery in that notorious *cul-de-sac* of embryology, the reduction of chromosomes, which is by many mistakenly supposed to be a veritable gold mine, the book contains practically no references to the work and advances of the last four or five years. A morphological continuity of germ-cells as the basis of heredity is still as strange and foreign to Weismann as it was nearly twenty years ago (1885) when he denied its existence. This continuity, though often mistakenly supposed to be so, is not at all the same thing as the continuity of a germ-plasm. The latter is hypothetical and intangible; the former, the germ-cells, are actual living entities or organisms. Still no attempt is made to tackle with and explain the Mendelian results, and recent researches in this field are only briefly mentioned in footnotes.<sup>1</sup> Still more remarkable, even inexplicable, is the absence of all reference to Vol. ii. of "Die Mutationstheorie" of de Vries (1903).

But space does not permit of detailed examination. Indications were not wanting, in the first edition, that Weismann really intended his words to be taken literally, that the "Vortraege" were indeed to be his legacy to posterity. This is confirmed by the second edition, which to all intents is an unaltered reprint of that of 1902. To every ardent worker in science sooner or later there comes a time when he thirsts for the green fields, the babbling brook, and the delights of the flower-garden. With the zoologist this longing is innate, and often it haunts him through all the years of his working life. Johannes Mueller sighed for, but never achieved, the rustic cottage and garden on the banks of the Rhine; to others, such as Huxley, Owen, and Semper, fate has been kinder, and has permitted a natural enjoyment of such closing days of well-spent lives. If this work be really the prelude of Weismann's departure from the field of scientific research; while deploring this,

<sup>1</sup> On the other hand, with more zeal than discretion, some of his disciples see in the Mendelian results a confirmation of their views of the reduction of chromosomes by means of a "reducing division." There could only be truth in this were Mendelian results universal in bi-sexual reproduction. It is, however, well known that this is by no means the case.

let it be recognised that his labours, to be witnesses of which has been our privilege, were those of a giant, let us wish him in his retirement from the field of strife many years of contented enjoyment of those beauties of animate Nature which he knows so well to appreciate and feel. But let not the reminder be lacking, "that a life is all too little to complete a cherished plan." No more than the Darwinian theory may those of Weismann, for they are many, be regarded as other than transient phases in the slow, sure, unfaltering advance of science. Though in a sense its premisses be sound, the Darwinian theory of natural selection explains nothing at all, and, on the other hand, Weismann's theories, while they would account for almost everything but the nature of life itself, unfortunately are founded more in the imagination than in actual facts of science. In addition to the "biophores," etc., and "germinal selection," other main supports of Weismann's theories are direct development, recapitulation, and a continuity of a germ-plasm. Nature, on the other hand, employs antithetic alternation of generations and a continuity of germ-cells, while she dispenses entirely with recapitulation. In the past forty years in many directions it has been the happy lot of Weismann "to close a pathway leading to error," as Huxley was wont to say; and with much solid discovery and investigation his honoured name is linked for all time. Of many scientific workers Nature is envious: to him she has been generous, and has given of her choicest secrets. More than has fallen to Weismann to accomplish is granted to few.

J. BEARD.

# Bibliography

## ANATOMY

- W. RUBASCHKIN. Studien über Neuroglia. *Arch. f. mikros. Anat.*, Bd. lxiiv H. 4, 1904, S. 575.  
 SCHÜLLER. Die Schadelbasis in Röntgenbild. Gräfe & Sillem, Hamburg, 1904, M. 14.  
 MARIE A. GREENE. Topographical Anatomy of the Human Head as shown by a Series of 11 Drawings of Cross Sections. *Journ. Kansas Med. Soc.*, Sept. 1904.

## COMPARATIVE ANATOMY—

- AUG. GIERSE. Untersuchungen über das Gehirn und die Kopfnerven von *Cyclothone acclindensis*. *Gegenbaur's Morphol. Jahrb.*, Bd. xxxii., H. 4, 1904, S. 602.  
 O. CHARNOCK BRADLEY. The Mammalian Cerebellum: Its Lobes and Fissures. *Journ. Anat. and Physiol.*, Vol. xxxix., 1904, p. 99.

## PHYSIOLOGY

- E. BAGLIONI. La Fisiologia del midollo spinale isolato. *Ztschr. f. allg. Physiol.*, Bd. iv., H. 2 u. 3, 1904, S. 384.  
 ALFRED W. CAMPBELL. Histological Studies on the Localisation of Cerebral Function. *Journ. Ment. Sc.*, Oct. 1904, p. 651.  
 BENAKY. Contribution à l'étude des fonctions de la couche optique. *Arch. de Neurol.*, Nov. 1904, p. 385.  
 T. GREGOR BRODIE and W. D. HALLIBURTON. Heat Contraction in Nerve. *Journ. of Physiol.*, Nov. 2, 1904, p. 473.  
 G. A. PARL. Sull' adattamento dell' eccitabilità dei centri nervosi e sui rapporti con la legge di Weber. *Ztschr. f. allg. Physiol.*, Bd. iv., H. 2 u. 3, 1904, S. 215.

## PSYCHOLOGY

- HANS BERGER. Ueber die körperlichen Ausserungen psychischer Zustände. *Atlas*. Gustav Fischer, Jena, 1904.  
 TREITEL. Haben kleine Kinder Begriffe? *Arch. f. d. gesam. Psychol.*, Bd. iii., H. 4, 1904, S. 341.  
 P. HOSPITAL. D'une maladie de l'attention. *Ann. méd-psychol.*, nov.-déc. 1904, p. 379.  
 BIAUTE. Des Maladies du sommeil et des crimes commis dans le somnambulisme. *Ann. méd-psychol.*, nov.-déc. 1904, p. 399.  
 JOS. FRÖBES. Ein Beitrag über die sogenannten Vergleichenungen übermerklicher Empfindungsunterschiede. *Ztschr. f. Psychol. u. Physiol. d. Sinnesorgane*, Bd. xxxvi., H. 4, 1904, S. 241.  
 G. A. HOEFER. Untersuchungen über die akustische Unterschiedempfindlichkeit des Weber-Fechnerschen Gesetzes bei normalen Zuständen, Psychosen und funktionellen Neurosen. *Ztschr. f. Psychol. u. Physiol. d. Sinnesorgane*, Bd. xxxvi., H. 4, 1904, S. 269.  
 HAHN. Ueber sinnvolles Verlesen. (Antwort an die Erwiderung von Jung.) *Arch. f. d. gesam. Psychol.*, Bd. iii., H. 4, 1904, S. 351.  
 PETERS. Die Farbenempfindung der Netzhautperipherie und konstanter subjektiver Helligkeit. *Arch. f. d. gesam. Psychol.*, Bd. iii., H. 4, 1904, S. 354.

- ADAMKIEWICZ. Über das unbewusste Denken und das Gedankensehen. Braumüller, Wien, 1904, M. 1.20.  
 MARTIAL VIGNOLLE. Les sauveteurs. Étude psychologique. *Ann. méd.-psychol.*, nov.-déc., 1904, p. 369.  
 L. WAHL. Un neurologue du grand siècle François Sylvius Deloboë, disciple de Descartes. *Ann. méd.-psychol.*, nov.-déc., 1904, p. 353.

## PATHOLOGY

- LUNDBORG. Spielen die Glandulae parathyreoideae in der menschlichen Pathologie eine Rolle? *Deutsche Ztschr. f. Nervenheilk.*, Bd. xxiv., H. 3 u. 4, 1904, S. 217.  
 CATOLA. Étude clinique et anatomo-pathologique sur les lacunes de désintégration cérébrale. *Rev. de méd.*, oct. 10, 1904, p. 778.  
 BURZIO. Sulle alterazioni delle fibre nervose spinali e dei gangli intervertebrali in alcune forme di psicosi croniche. (Cont. e fine.) *Ann. di Freniatria*, Vol. xiv., f. 8, 1904, p. 198.

## CLINICAL NEUROLOGY AND PSYCHIATRY

### GENERAL—

- H. OPPENHEIM. Lehrbuch der Nervenkrankheiten für Ärzte und Studierende. Vierte Auflage. S. Karger, Berlin, 1906, 30/.  
 FRANK R. FRY. President's Address at the Thirtieth Annual Meeting of the American Neurological Association. *Journ. Nerv. and Ment. Dis.*, Nov. 1904, p. 689.  
 ANTON. Ueber Nerven- und Geisteskrankung in der Zeit der Geschlechtsreife. *Wien. klin. Wchschr.*, Nov. 3, 1904, S. 1161.  
 P. M. KING. Report of Injuries of the Brain and Cord. *Carolina Med. Journ.*, Sept. 1904.  
 EDINGER. Die Aufbauchkrankheiten des Nervensystems. *Deutsche med. Wchschr.*, Nov. 3, 1904, S. 1633.  
 JAMES TAYLOR. A Lecture on Paralysis as it occurs in Children. *Lancet*, Nov. 12, 1904, p. 1836.  
 HELDENBERG. Névrosologie et névroso-pathologie. *Journ. de Neurol.*, oct. 20, 1904, p. 381.  
 LICHTHEIM. Nekrolog Carl Weigert. *Deutsche Ztschr. f. Nervenheilk.*, Bd. xxvii., H. 8 u. 4, 1904, S. 840.

### MUSCLES—

- P. SIMON et TH. GUILLOZ. Un cas de myopathie primitive progressive. *Res. méd. de l'Est*, août 1904.  
 JOHN HILL ABRAM. Inflammation of the Muscles, with Special Reference to Two Cases of Infective Myositis. *Lancet*, Nov. 12, 1904, p. 1841.  
 PRĘBOBRAGENSKI et MARGOULISS. Contribution à l'étude de la polymyosite. *Rev. de méd.*, oct. 10, 1904, p. 765.  
 Thomsen's Disease.—FRANKL-HOCHWART. Die Thomsen'sche Krankheit. *Deutsche Klinik*, Bd. vi., Abt. 1, 1904, S. 764.

### PERIPHERAL NERVES—

- SEIFFER. Ueber zwei seltene Fälle peripherer Nervenlähmung. *Monatsschr. f. Psychiat. u. Neurol.*, Bd. xvi., H. 4, 1904, S. 425.  
 EVRARD. Des polynévrites blennorragiques. Roussel, Paris, 1904.  
 J. D. ROLLESTON. Clinical Observations on Diphtheritic Paralysis. *Practitioner*, Nov. 1904, p. 597.  
 HENRY E. TULEY. Acute Ascending or Landry's Paralysis; Report of a Case in a child of two years. *Kentucky Med. Journ.*, Sept. 1904.

### SPINAL CORD—

- Meningitis.—OSKAR FISCHER. Zur Frage der Pachymeningitis interna chronica cervicalis hyperplastica. *Ztschr. f. Heilk.*, Bd. xxv., H. x., 1904, S. 339.

- VAISSIER et LEDOUX.** Un cas de méningite oébrospinale à méningocoques. Dodivert, Besançon, 1904.
- ALFRED FUCHS** and **R. ROSENTHAL.** Physikalisch-chemische zytologische und anderweitige Untersuchungen der Zerebrospinalflüssigkeit. *Wien. med. Press.*, Okt. 30 u. Nov. 6, 1904, S. 2082.
- Tabes.**—**J. F. DONLEY.** On the Early Diagnosis of Tabes Dorsalis. With Report of a Case. *Providence Med. Journ.*, Sept. 1904.
- LEONARD J. KIDD.** A Fatal Case of Tabetic Gastric Crisis. *Lancet*, Nov. 5, 1904, p. 1282.
- Lateral Sclerosis.**—**STRÜMPPELL.** Die primäre Seitenstrangaklerose (spastische Spinalparalyse). *Deutsche Zechr. f. Nervenheilk.*, Bd. xxvii., H. 3 u. 4, 1904, S. 291.
- Poliomyelitis.**—**BRUINING.** Zwei Fälle von sog. Poliomyelitis anterior chronica bei Vater und Sohn. *Deutsche Zechr. f. Nervenheilk.*, Bd. xxvii., H. 3 u. 4, 1904 S. 269.
- Myelitis.**—**JOSEPH COLLINS.** Purulent Myelitis, Focal and Disseminated, *Journ. Nerv. and Ment. Dis.*, Nov. 1904, p. 695.
- Disseminated Sclerosis.**—**CASSIRER.** Die Sklerosis multiplex. Koenig, Leipzig, 1904.

#### BRAIN AND MEMBRANES—

- BARDET.** Les lésions oébrales dans la mort apparante du nouveau-né. *Thèse de Paris*, 1904.
- ADOLPH BRÖNNER.** Notes on a Case of Thrombosis of the Cavernous Sinus due to Empyema of the Sphenoidal Sinus. *Brit. Med. Journ.*, Nov. 12, 1904, p. 1310.
- PIERRE MARIE** et **IDLESOHN.** Un cas de lésion linéaire limitée à la substance blanche de la frontale ascendante droite dans sa moitié supérieure. Hémiplegie. Étude de la dégénération secondaire pyramidale. *Rev. Neurol.*, Oct. 30, 1904, p. 1026.
- A. PICK.** Zur Symptomatologie der linksseitigen Schlafenlappenatrophie. *Monatschr. f. Psychiat. u. Neurol.*, Bd. xvi., H. 4, 1904, S. 378.
- G. B. HASSIN.** Hemiplegia Progressiva Ascendens Acuta. (Report of a Case.) *Clin. Review*, Chicago, Sept. 1904.
- Meningitis.**—**R. BENSAUDE** et **L. RIVET.** Un cas de méningite aiguë syphilitique. *Arch. gén. de méd.*, Nov. 1, 1904, p. 2766.
- Bulbar Paralysis.**—**HENNEBERG.** Ueber einen mit Bulbärparalyse komplizierten Fall von Systemerkrankung. *Monatschr. f. Psychiat. u. Neurol.*, Bd. xvi., H. 4, 1904, S. 409.
- Tumeur.**—**NONNE.** Über Fälle von Symptomencomplex "Tumor Cerebri" mit Ausgang in Heilung (Pseudotumor cerebri). Über letal verlaufene Fälle von "Pseudotumor cerebri" mit Sektionsbefund. *Deutsche Zechr. f. Nervenheilk.*, Bd. xxvii., H. 3 u. 4, 1904, S. 169.
- Abscess.**—**UCHERMANN.** Otitic Brain Abscess. *Arch. of Otol.*, Vol. xxxiii., No. 5, 1904, p. 416.
- Aneurism.**—**ALEXANDER BRUCE** and **W. B. DRUMMOND.** A Case of Intracranial Aneurism in a Young Subject. *Rev. Neurol. and Psychiat.*, Nov. 1904, p. 737.

#### MENTAL DISEASES—

- J. T. SEARCY.** Mental Abnormalities. *Alabama Med. Journ.*, Sept. 1904.
- W. H. B. STODDART.** The Psychology of Hallucination. *Journ. Ment. Sc.*, Oct. 1904, p. 633.
- WILLIAM A. WHITE.** Hallucinations. *Journ. Nerv. and Ment. Dis.*, Nov. 1904, p. 707.
- GERSHOM H. HILL.** Delirium. *Iowa Med. Journ.*, Sept. 15, 1904.
- SKLIAR.** Ueber Gefängnispsychosen. *Monatschr. f. Psychiat. u. Neurol.* Bd. xvi., H. 4, 1904, S. 441.
- R. PERCY SMITH.** The Presidential Address on Paranoia, delivered at the Sixty-third Annual Meeting of the Medico-Psychological Association, held in London, July 1904. *Journ. Ment. Sc.*, Oct. 1904, p. 607.

- ALBERT WILSON. A Case of Double Consciousness. *Journ. Ment. Sc.*, Oct. 1904, p. 699.
- J. E. DONLEY. Three Cases of the Association Neurosis, with Remarks on its Genesis. *Boston Med. and Sur. Journ.*, Nov. 3, 1904, p. 484.
- C. A. MERCIER. Stress Again. *Journ. Ment. Sc.*, Oct. 1904, p. 697.
- OLIVA. Due casi di inversione sessuale. (Cont. e fine.) *Ann. di Freniatria*, Vol. xiv., f. 3, 1904, p. 255.
- ROBERT H. GRUBB. The Plea of Insanity. *Cincinnati Lancet-Clinic*, Sept. 24, 1904.
- S. TÜRKEL. Die kriminellen Geisteskranken. M. Perles, Wien, 1904, M. 2.
- C. A. MERCIER. Statistical Tables. *Journ. Ment. Sc.*, Oct. 1904, p. 672.
- DORNBLÜTH. Moderne Einteilung der Geisteskrankheiten. *Munch. med. Wchnschr.*, Nov. 1, 1904, S. 1938.
- W. A. POTTS. The Problem of the Morally Defective. *Lancet*, Oct. 29, 1904, p. 1210.
- FERDINAND KLEIN. Therapeutische Erfahrungen bei akuten Psychosen. *Monatschr. f. Psychiat. u. Neurol.*, Bd. xvi., H. 4, 1904, S. 382.

#### GENERAL AND FUNCTIONAL DISEASES—

- DECROLY. Contribution à la symptomatologie du spasme salutatoire (epilepsia nutans). *Journ. de Neurol.*, Oct. 20, 1904, p. 890.
- WERNICKE. Ein Fall von Crampus-Neurose. *Berl. klin. Wchnschr.*, Okt. 24, 1904, S. 1121.
- Chorea.**—FLATAU. Die Chorea. Koenig, Leipzig, 1904.
- OTFRID FÖRSTER. Das Wesen der choreatischen Bewegungstörung. *Samm. klin. Vorträge*, Nr. 332, 1904, S. 259.
- Paralysis Agitans.**—BRUNS. Zur Symptomatologie der Paralysis agitans. *Neurol. Centralbl.*, Nov. 1, 1904, S. 878.
- Epilepsy.**—A. PARIS. Epilepsie: pathogénie et indications thérapeutiques. *Arch. de Neurol.*, Nov. 1904, p. 369.
- WILLIAM O'NEILL. A Case of Attacks of Temporary Loss of Consciousness and Memory. *Lancet*, Nov. 12, 1904, p. 1345.
- H. K. ABBOTT. A Case of Epilepsy with Necrotic Softening of the Brain due to Injury. *Lancet*, Nov. 12, 1904, p. 1346.
- Neurasthenia.**—MAURICE BOIGEY. La neurasthénie dans l'armée. *Rev. Neurol.*, oct. 30, 1904, p. 1030.
- E. CUTTER. Male Neurasthenia. *Virginia Med. Semi-Monthly*, Sept. 23, 1904.
- F. E. FRANCHERE. The Ocular Manifestations of Neurasthenia. *Med. Herald*, St Joseph, Sept. 1904.
- BOSMA. Nervöse Kinder. J. Ricker, Giessen, 1904, M. 1.60.
- Hysteria.**—OSKAR WOLTÄR. Wandertrieb bei einer Hysterischen. *Prag. med. Wchnschr.*, Nov. 3, 1904, S. 565.
- J. S. MACKINTOSH. A Case of Hysteria Major: the Nemesis of "Dowicizm"! *Lancet*, Nov. 5, 1904, p. 1280.
- JUNG. Über hysterisches Verlesen. Eine Erwiderung an Herrn Hahn. *Arch. f. d. gesam. Psychol.*, Bd. iii., H. 4, 1904, S. 347.
- COTTEU. L'Hystéro-Traumatisme cardiaque. Rousset, Paris, 1904.
- Tetany.**—CURSCHMANN. Tetanie, Pseudotetanie und ihre Mischformen bei Hysterie. *Deutsche Ztschr. f. Nervenheilk.*, Bd. xxvii., H. 3 u. 4, 1904, S. 239.
- Migraine.**—L. HARRISON METTLER. Hemicrania Migraine. *Clin. Review*, Chicago, Sept. 1904.
- Exophthalmic Goitre.**—ERWIN MIESOWICZ. Ein Fall von Basedow'scher Krankheit mit Muskelatrophien und sekretorischer Insuffizienz der Magenschleimhaut. *Wien. klin. Wchnschr.*, Nov. 10, 1904, S. 1206.
- JELLINEK. Ein bisher nicht beachtetes Symptom der Basedow'schen Krankheit. *Wien. klin. Wchnschr.*, Okt. 27, 1904, S. 1145.
- EULENBURG. Die Basedow'sche Krankheit nach ihrem heutigen Stande in Theorie und Praxis. *Deutsche Klinik*, Bd. vi., Abt. 1, 1904, S. 744.



## ALCOHOLISM, POISONS, ETC.—

- ZIEHEN. Über den Einfluss des Alkohols auf das Nervensystem. *Mässigkeits-Verlag*, Berlin, 1904, M.—20.  
 J. W. DUNNING. The Inebriate. A Medical and Social Problem. *St Paul Med. Journ.*, Oct. 1904.  
 J. M. CATCHING. Treatment of the Opium Habit by Hyoscin Hydrobromate. *Journ. Mississippi State Med. Assoc.*, Sept. 1904.

## SPECIAL SENSES AND CRANIAL NERVES—

- POULTON. The Vision of Birds. *Brit. Med. Journ.*, Nov. 12, 1904, p. 1287.  
 W. H. RIVERS. Investigations on the Comparative Visual Acuity of Savages and of Civilised People. *Brit. Med. Journ.*, Nov. 12, 1904, p. 1297.  
 BUMKE. Untersuchungen über den galvanischen Lichtreflex. *Ztschr. f. Psychol. u. Physiol. d. Sinnesorgane*, Bd. xxxvi, H. 4, 1904, S. 294.  
 KARL KRONER. Ueber Gesichtsfeldermüdung. *Ztschr. f. klin. Med.*, Bd. liv., H. 3 u. 4, 1904, S. 265.  
 ONODI. The Disturbances of Vision and Development of Blindness of Nasal Origin Induced by Disease of the Posterior Accessory Sinuses. *Brit. Med. Journ.*, Nov. 5, 1904, p. 1212.  
 C. A. VEASEY. Report of Two Cases of Family Macular Degeneration of the Cornea. *Arch. of Ophth.*, Bd. xxxiii, Nov. 5, 1904, S. 510.  
 R. MARCUS GUNN and Others. Discussion on Retro-ocular Neuritis. *Brit. Med. Journ.*, Nov. 12, 1904, p. 1285.  
 HAMLIN and W. C. POSEY. Associated Movements of the Head and Eyes in Infants. *Am. Journ. Med. Sc.*, Nov. 1904, p. 875.  
 LUCIEN HOWE. On the Measurement of the Rapidity of the Lateral Motion of the Eye. *Brit. Med. Journ.*, Nov. 12, 1904, p. 1203.  
 BUMKE. Die Pupillenstörungen bei Geistes- und Nervenkrankheiten. *Fischer*, Jena, 1904, M. 5.  
 VOLLERT. Ein Fall von Lähmung des rechten Obliquus superior mit kontralateralen Paraesthesien der linken Gesichtshälfte, erworbenem cerebralen Nystagmus und Schwindelerscheinungen. Heilung. *Munch. med. Wchnschr.*, Nov. 8, 1904, S. 2001.  
 H. M. SHERMAN. Intracranial Neurectomy for Trigeminal Neuralgia. Cases and Comments. *Journ. Am. Med. Assoc.*, Oct. 1904, p. 941.  
 CHAS. H. FRAZIER and W. G. SPILLER. Physiological Extirpation of the Ganglion of Gasser. Further Note on Division of the Sensory Root for the Douleureux. *Journ. Am. Med. Assoc.*, Oct. 1904, p. 943.  
 G. MIRTO. Spasmodico dell' elevator della palpebra superiore guarito mediante le applicazioni polari anodiche. *Gion. di elettricità med.*, Vol. iv., Nr. 1, 1904.  
 W. MILLIGAN. Ménière's Disease: a Clinical and Experimental Enquiry. *Brit. Med. Journ.*, Nov. 5, 1904, p. 1228.  
 W. S. BRYANT. A Phonographic Acoumeter. *Arch. of Otol.*, Vol. xxxiii, No. 5, 1904, p. 688.  
 SIEBENMANN. A Case of Pulmonary Tuberculosis, with Interstitial Neuritis of both Cochlear Nerves, together with Persistent Embryonic Adhesions in the Scala Tympani. *Arch. of Otol.*, Vol. xxxiii, No. 5, 1904, p. 368.

## MISCELLANEOUS SYMPTOMS—

- LEONARD J. KIDD. The Mode of Turning, in Walking, of Organic Hemiplegics and Spastic Paraplegics. *Rev. Neurol. and Psychiat.*, Nov. 1904, p. 742.  
 J. HOGARTH PRINGLE. Collateral Paralysis the Result of Cerebral Trauma. *Scot. Med. and Surg. Journ.*, Nov. 1904, p. 394.  
 FRANKL-HOCHWART. Ueber Athetose. *Deutsche Klinik*, Bd. vi., Abt. 1, 1904, S. 709.  
 VICTOR HORSLEY. On Tactile Sensation. *Practitioner*, Nov. 1904, p. 581.  
 HUGO LEVI. Beitrag zur Casuistik der Halsrippen. *Neurol. Centralbl.*, Nov. 1, 1904, S. 988.  
 HENRY KOPLIK. Percussion of the Skull with Especial Reference to the Ocular Occurrence of M'Ewen's Sign in Meningitis and Cerebral Disease of Children. *Arch. of Pediatrics*, N.Y., Sept. 1904.  
 ROGER S. MORRIS. Angio-Neurotic Oedema. *Am. Journ. Med. Sc.*, Nov. 1904, p. 612.

- CASSIRER. Die vasomotorisch-tropischen Neurosen. *Deutsche Klinik*, Bd. vi., Abt. 1, 1904, S. 719.
- TESTI. Edema neurotrofico e vasomotorio dell' arto superiore destro. *Riv. crit. di clin. med.*, Ott. 22 e 29, 1904, p. 685 e 701.
- STADELMANN. Die Katatonie. Ballhorn & Cramer, Würzburg, 1904, M. 3.
- A. PICK. Studien über motorische Apraxie und ihr nahestehende Erscheinungen. F. Deuticke, Wien, 1904, M. 7.
- HINSHELWOOD, MACPHAIL and FERGUSON. A Case of Word-Blindness, with Right Homonymous Hemianopsia. *Brit. Med. Journ.*, Nov. 12, 1904, p. 1304.
- JAMES HINSHELWOOD. A Case of Congenital Word-Blindness. *Brit. Med. Journ.*, Nov. 12, 1904, p. 1303.
- W. S. SYME. A Case of Congenital Word-Deafness. *Brit. Med. Journ.*, Nov. 5, 1904, p. 1229.

## TREATMENT—

- G. E. SHUTTLEWORTH. The Educational Treatment of Young Epileptics. *Journ. Ment. Sc.*, Oct. 1904, p. 606.
- J. H. POTTER. Spasmodic Neuralgia Treated with Bromide. *Brit. Med. Journ.*, Oct. 29, 1904, p. 1170.
- CHIPAULT. Chirurgie nerveuse d'urgence. Baillière et fils, Paris, 1904, 1 fr. 50.
- FAURE. Le traitement chirurgical de la paralysie faciale. *Presse méd.*, Nov. 5, 1904, p. 707.
- J. C. MUNRO. Laminectomy. *Journ. Am. Med. Assoc.*, Oct. 23, 1904, S. 1183.
- DENKER. On the Operative Treatment of Otitic Intracranial Complications. *Arch. of Otol.*, Vol. xxxiii., No. 5, 1904, p. 409.
- FRAENKEL, RAMSEY HUNT, WOOLSEY and ELSBERG. Contribution to the Surgery of Neurofibroma of the Acoustic Nerve. *Ann. of Surg.*, Phila., Sept. 1904.
- DEBRITTO. Contribution à l'étude de la névrectomie opticooculaire. *Clinique ophtalmol.*, août 25, 1904, p. 267.
- A. H. TUBBY. A Case illustrating the Operative Treatment of Paralysis of the Serratus Magnus by Muscle Grafting. *Brit. Med. Journ.*, Oct. 29, 1904, p. 1159.

# Indices

*Page references to Original Articles are indicated by heavy type figures.*

## SUBJECT INDEX

- ABERRANT** Circumolivary Bundle, 377  
**Achondroplasia**, Alexia in, 411  
**Acoustic Neuromata**, 603  
**Acromegaly**, 466  
**Acroparæsthesia**, Objective Sensory Disturbances in, 323  
**Adiposia Dolorosa**, 713, 771  
**Agraphia**, with Verbal Blindness, 658  
**Alalia**, Coën's Idiopathic, 471  
**Albany Hospital Report**, 485  
**Alcoholism**: Social Causes of, 714; Relation to Insanity, 716; Pathology of, 403; Corpus Callosum in, 153; Clinical Forms of Chronic, 328; Continuous Delirium, 325; Delirium of Magnan, 662; Well-to-do Inebriate, 85; Treatment, 716; "Gold Cure" in, 561; Atropin in, 562; Relation to Arteriosclerosis, 833  
**Alexia**, 321; in Achondroplasia and Imbecility, 411; and Agraphia, 658; Word-Blindness, 827; Congenital; 829  
**Amaurosis**: Retina in Tabetic, 755  
**Amaurotic Family Idiocy**, 250  
**Amentia**, relation to Epilepsy, 604  
**Amnesia**, in Syphilis, of Nervous System, 764  
**Amnesia verbalis**, *vide* Alexia  
**Anæsthesia**, Spinal, in Labour, 845  
**Anæsthesia**, Visceral, in General Paralysis, 470  
**Anencephaly**, 152  
**Aneurism**, Intra-Cranial, 737; Cirroid, of Cervical Cord, 545  
**Ankle Clonus**, 85  
**Anomia**, *vide* Aphasia  
**Anophthalmus**, Congenital Bilateral, 456  
**Anti-Serums** in Acute Insanity, 483  
**Aphasia**: with Sensory Anomia, 606; Verbal Blindness with Agraphia, 658; Sensory Aphasia, 711; Congenital Word Blindness, 711; with left-sided Hemiplegia in Right-handed Individuals, 553; Dysantigraphia, 323, *vide* Alexia; 826  
**Apoplexy**, Juvenile, 708  
**Arterio-sclerosis**: Relation of Alcohol to, 833  
**Astereognosis**: Functional, 579; in Cerebral Affections, 470  
**Ataxy**, Hereditary Cerebellar, Pathological Anatomy of, 756; Cerebellar Hemiataxia, 661; *vide* Friedreich's Ataxy, Tabes, etc.  
**Atrophy of Muscles of Thigh and Leg**, treated by Sinusoidal Voltaisation, 831  
**Atrophy**, Muscular, caused by Encephalomyelitis, 34  
**Auditory Nerve**, Tumour of, 77; Acoustic Neuromata, 603  
**Auricular Vertigo**, Lumbar Puncture in, 334  
**Auto-Intoxication** in Epilepsy, 319  
**BABINSKI'S Sign**, *vide* Reflexes  
**Basal Opticus Root**, 386; Tractus Peduncularis Transversus, 386  
**Basilar Meningitis**, 403, 459  
**Benedict**, Inferior Syndrome of, 156  
**Beri-beri** in Monkeys, 409  
**Blood**: in Dementia Paralytica, 154; in Epilepsy, 585; in Mental Disease, 666  
**Blood-vessels**, Innervation of Pulmonary, 295  
**Brachial Plexus**: its Motor Distribution, 688  
**Brain**: *Anatomy*: Collateral Sulcus, 746; "Affenspalte," 55; Nucleus Lateral to Fasciculus Solitarius, 55; Parieto-occipital Fissure, 217; Fossa Parieto-occipitalis, 218; "Limbus Postorbitalis," 142; "Transitory Fissures" of, 442; "Fissura Perpendicularis Externa," 442; Occipital Region, 443; Ventral Tegmentum Region, and Median Fillet, 687; Funiculus Cuneatus, 687; Circumolivary

- Bundle, 377; Deiters' Nucleus, 383; Morphology of Egyptian, 443; Apparatus for Estimating Volume of, 746; Retrocalcarine Cortex, 442; Pedunculus Corporis Mammillaris, 385; Ganglion Tegmenti Profundum, 385; Dorso-Ventral Raphe, Fibres of Tegmentum, 385; Structure of Pons, 687; Sulci of Brains of Australian Aborigines, 218  
*Comparative Anatomy*: Morphology of the Ape's Brain, 442; Variations in Cerebral Fissures in Apes, 442; Occipital Lobes, 442; Occipital Region of Cerebral Hemisphere in Man and Apes, 443; Association Systems of Mammalian Brains, 448; Mammalian Cerebellum, 744; Neuromeres of Rhombencephalon of Pig, 635  
*Physiology*: True Motor Centres, 584; Areas and Centres of Cerebral Cortex, 747; Projection of Retina on Calcarine Cortex, 75; Contribution to Physiology of Central Nervous System, 152; Effect of Electricity on Cerebral Pulse, 591; Coloured Audition, 302; Cortical Localisation of Vision, 820; Cerebral Zone of Speech, 826  
*Pathology*: Pithecooid Abnormality of Sylvian Region, 219; Anophthalmus, 456; Porencephalic, 406; of Microcephalic Idiot, 232; Aneurism of, 737; Parasitology of, 701; Corpus Callosum in Alcoholic Subjects, 153; Bilateral Loss of Post-central Cortex, 754; in Cretins, 457; *vide* Cerebellum, Medulla, etc.  
 Brown-Sequard's Paralysis, 547  
 Buccal Perforation in Tabes, 70  
 Bulbar Paralysis; Post-Diphtheritic Chronic, 461, 652; Apoplecticiform, 316; Hemorrhage, 155; *vide* Myasthenia Gravis  
**CALCARINE Cortex**, Projection of Retina on, 75  
 Catatonia, 329, 330  
 Cauda Equina, Tumour of, 233  
 Central Nervous System, Physiology of, 152  
 Cephalopathy, Acute Diffuse Parenchymatous, 458  
 Cerebellar Ataxy, Pathological Anatomy of, 756  
 Cerebellum: Defective Development of Lateral Cerebellar Lobes in Dog, 755; Cerebellar Localisation, 585  
 Cerebral Degeneration with Symmetrical Changes in Macula, 242  
 Cerebral Pulse, Effect of Electricity on, 591  
 Cerebro-Spinal Fluid: in Nervous Diseases, 401; in Mental Diseases, 537; in Tabes, 539; in General Paralysis, 540; in Syphilis, 540; in Meningitis, 107; Lymphocytosis of, 402; Cytological Examination of, 651; Choline in, in Epilepsy, 62, 651; Choline in, as Sign of Nervous Tissue Degeneration, 651; Phosphoric Acid in, 652  
 Cerebro-Spinal Meningitis, *vide* Meningitis  
 Cervical Ribs, 830  
 Choked Disc, *vide* Optic Neuritis  
 Choline: in Cerebro-Spinal Fluid in Epilepsy, 62, 651; as Sign of Nervous Tissue Degeneration, 651  
 Chorea: Localisation of Hemichorea, 236  
 Circumolivary Bundle, 377  
 Cheiromegalia with Syringomyelia, 69  
 Cirroid Aneurism of Cervical Cord, 545  
 Claudication, Intermittent, 81 (2)  
 Clonus, *vide* Reflexes  
 Coloured Audition, 302  
 Combined Sclerosis, 431, 616, 648; Tabetic, 458  
 Compression-Myelitis, 704, *vide* Spinal Cord  
 Consciousness, Evolution of, 151  
 Corneal Erosion, Recurrent, and Trigeminal Neuralgia, 556  
 Corneo-mandibular Reflex, 163 (3)  
 Corpora Quadrigemina, Cells of, 529  
 Corpus Callosum: Absence of, in Microcephalic Brain, 232; in Alcoholic Subjects, 153  
 Cortex Cerebri, Action of Radium on, 396, *vide* Brain  
 Craniectomy by Means of Gigli's Saw, 486  
 Cremasteric Reflex, 555  
 Cretins, Central Nervous System in, 457  
 Crossed Aphasia, 553  
 Crural, Paralysis of Anterior, 409  
 Currents of High Frequency and High Tension in "Maladies par Ralentissement de la Nutrition," 484  
 Cysts, Congenital, of Fourth Ventricle, 592  
 Cytodiagnosis, *vide* Cerebro-Spinal Fluid  
**DRAF-MUTISM**, 471  
 Degeneracy and Degenerate, with Plea for Supplementary Terms Deviation and Deviate, 252  
 Degenerations: of Nervous Tissue, 651; from Lesions of Posterior Nerve Roots, 450; from Transverse Lesions of Spinal Cord, 450; Experimental Secondary, studied by Donaggio's Method, 811; Positive Coloration of Nerve Fibres in, 812; Mechanism of Reaction of, 832

Deiters' Nucleus, Central Connections and Neighbouring Nuclei, 383  
 Delirium, Pathological Anatomy of Acute, 458  
 Delusions in General Paralysis, Pathogenesis of, 332; of Persecution in Hypochondriasis, 477  
 Dementia, with Multiple Sclerosis, 598  
 Dementia Paralytica, *vide* General Paralysis  
 Dementia Præcox, 610, 719; Catatonic, 781  
 Dementia, Senile, 478  
 Deroum's Disease: *vide* Adiposis Dolorosa  
 Deviation and Deviate: a Plea for Introduction of Terms, 252  
 Diabetic Neuritis, 645  
 Diaphragm, Respiratory Movements of, 150  
 Diphtheritic Paralysis, 652, 461  
 Diplegia, Reflex Movements in, 84; Amaurotic Family Idiocy, 250; Facial Diplegia, 320  
 Diplococcus of Weichselbaum, Meningitis due to, 311  
 Dispensary Work in Nervous and Mental Diseases, 537  
 Disseminated Sclerosis, 497, 546, 710; Frequency and Etiology, 462; Occurrence and Etiology, 597, 649; with Dementia, 598; Family, 758; Histology of, 304; Non-myelinated Nerve Fibres in Foci of, 304; Remissions and Relapses in, 818  
 Dysantigraphia, 322  
 Dysarthritic Trouble in Epileptic Child, 237  
 ECLAMPSIA, Thyroid Extract in, 335  
 Ego, Psychological and Psychiatric Contributions to Study of, 398  
 Electricity, Effect on Cerebral Pulse, 591  
 Electrical Treatment: Methods of Application of High Frequency Currents, 562; High Frequency Current, 484; In Atrophy of Muscles of Thigh and Right Leg, 831; Principles of, in Nervous Disease, 842; of Facial Neuralgia, 843; Oscillating Current in Sciatica, 844  
 Emphysema of Skin after Epileptic Attack, 238  
 Encephalitis, 599  
 Encephalomyelitis, Muscular Atrophy caused by, 34  
 Epilepsy: Blood in, 535; Choline in Cerebro-Spinal Fluid in, 62, 651; Auto-intoxication in, 819; Is Epilepsy a Functional Disease? 413; Pre-Epileptic

Stage, 766; Mental Condition in, 412; Subcutaneous Hemorrhage in, 552; Emphysema of Skin in, 238; Dysarthritic Trouble in 237; Status Epilepticus, 767; Relation to Amentia, 604; Uncinate Group of Fits, 552; Psycho-Epilepsy of Janet, 612; Relation to Life Assurance, 778; Serum Treatment, 482, 782; Dietetic Bromide Treatment, 253; Salt Starvation in Treatment, 793  
 Erb's Disease, *vide* Myasthenia Gravis  
 Ergographic Curve, 298, 590; Form of, 223  
 Ergotherapy and Psychotherapy 560  
 Erythromelalgia, 536; Its Relation to Raynaud's Symptom-Complex, 468  
 Exophthalmic Goitre, Serum Treatment of, 782  
 Eyeball, Movement of, accompanying Light Reaction, 162  
 Eyes: Tic of, 238; Congenital Bilateral Anophthalmus, 456; Innervation of Mammalian, 528; Muscles in Organic Hemiplegia, 265  
 FACE Spasm, 78, 240; Hemistrophy, 79; Hemihypertrophy, 80; Reflexes, 247, 274; Palpebral Symptoms, 163, 322; Orbicularis Oculi, 557; in Tetany, 654; Diplegia, 320; Surgical Treatment of Paralysis, 334  
 Family Nervous Disease, A Rare Form of, 705  
 Fat Pigment Granules in Central Nervous System, 389, 697  
 Fatigue, 255; Form of Ergographic Curve, 223; Influence on Reflexes, 297; Methods of Measuring, 456; Nervous Fatigue in Muscular Work, 395; Ergographic and Myographic Curve, 298, 590  
 Fever, Influence of, on Pains of Locomotor Ataxia, 538  
 Fibrillar Reticulum, *vide* Methods  
 "Fissura Perpendicularis Externa," 442  
 Friedrich's Ataxia, 541  
 GALLOPING Paralysis, 234  
 Ganglia: Endocellular Network in Nerve Elements of Spinal, 389; Cells of Sensory, 228; Spinal Root, in Tabes, 697; Bifurcation of Fibres in Spinal, 802  
 Ganser's Symptom, 607  
 Gastric Cancer, Tetany in, 656  
 General Paralysis: Cytodiagnosis in, 540; Blood in, 154; Cerebral Tumour with Clinical Appearance of, 551; Visceral Anæsthesia in, 470; Hypotonus in, 414; Pupils in, 318; Voice

- in, 72; Specific Delusions in, 332; Galloping Paralysis, 234; Taboparalysis in Childhood and Youth, 339; Relation between Syphilis and, 336; and Tabes, 71
- Giantism, Infantile Type of, 713
- Gigli's Saw, Craniectomy by Means of, 436
- Glottis, Respiratory Movements of, 394
- Gluteal Reflex, 714
- Gowers' Tract, Termination of, 685
- HÆMORRHAGE:** Subcutaneous, in Epilepsy, 552; Focal, in Bulb, 155
- Hallucinations of Hearing, 608
- Head: Noises in, 759; Involuntary Movements of, 324
- Headache, Neurasthenic, 607
- Hearing, Unilateral Hallucinations of, 608
- Heart, Nervous Affections of, 733; Nerves in Tabes, 167
- Hemianæsthesia: with Cerebral Hemiplegia, 600
- Hemianopia: Deviation of Eyes in Homonymous, 472; Immobility of Pupil in, 161; and Conjugate Deviation, 660; Quadrantic, 820; Right Homonymous, with Word-Blindness, 827
- Hemiataxia, Cerebellar, 661; and Infantile Cerebral Hemiplegia, 824
- Hemiatrophy, Progressive Facial, 79
- Hemicephaly, *vide* Anencephaly
- Hemichorea, Localisation of, 236
- Hemihypertrophy, Progressive Facial, 80
- Hemiedema in Hemiplegia, 777
- Hemiplegia: Mode of Turning in Walking in Organic, 742; Pain Sensation in Cerebral, 775; Hemiedema in, 777; Hemianæsthesia with, 600; Eye Muscles in, 235; Uræmic, 653; Tendon Reflexes in, 554; with Aphasia, 558; Movements of Superior Intercostal Muscles in, 160; Respiratory Movements in, 161; Infantile Cerebral, and Hemiataxia, 824; as Sign of Cerebral Tumour, 824
- Heredity and Variation, Morphological Continuity of Germ-Cells as Basis of, 1, 114, 185; from Psychiatric Aspect, 833; and Genetic Variation, 847
- Herpes Zoster, 816
- Hydrocephalus, Chronic Internal, 313, 759; Treatment by Auto-Drainage, 783
- Hydrophobia, Nervous System in, 405
- Hyoscine, Treatment of Morphine Habit by, 560
- Hypertonus, 775
- Hypertrophy: Hemihypertrophy of Face, 80
- Hypochondriasis with Delusions of Persecution, 477
- Hypotonus, 775; in General Paralysis, 414
- Hysteria: Diagnosis, 82; in Childhood, 157, 159; Metameric Manifestations in, 777; Isolation and Psychotherapeutics in, 668; Insanity, 611; Mental Affections (Review), 723; Hysterical Tetany, 772
- Hystero-traumatism, 155
- IDIOCY,** Anatomical Basis of, 698; Etiology of Simple, 475; Family Amaurotic, 250
- Impulsions and Obsessions, 85
- Infantile Paralysis, Etiology of Cerebral, 475
- Innervation of Pulmonary Blood-vessels, 295
- Insanity: Variation in Relation to Origin of, 235; Hysterical Insanity, 611, 723; Acute Hallucinatory, 87; Traumatic, 476; and Inebriety, 716; and Life Assurance, 778; Radio-active Bodies and, 781; Anti-serums in, 433; Metaphysical Conception of, 608
- Insular Sclerosis, *vide* Disseminated Sclerosis
- Intercostal Muscles in Hemiplegia, 160
- Intermittent Claudication, 81
- Involuntary Movements of Head, 324
- Isolation in Treatment of Neurasthenia and Hysteria, 668
- JANET,** Psycho-epilepsy of, 612
- Jolly, Professor Friedrich, Obituary Notice, 169
- KNEE-Jerk,** *vide* Reflexes
- Korsakoff's Symptom-Complex, 610; Differential Diagnosis, 245
- LEUCOCYTE** Counts in Mental Disease, 666
- Leprosy, Nervous, 773; or Syringomyelia? 650
- Levator Palpebræ Superioris, 163, 322
- Life Assurance, Insanity and Epilepsy in Relation to, 778
- Localisation: True Motor Centres, 584; Cerebellar, 585; Retina and Calcarine Cortex, 75
- Locomotor Ataxia, *vide* Tabes
- Lumbar Puncture: 39, 599, 650; in Hemorrhagic Fibrinous Meningitis, 459; in Psychiatry, 479; in Ear Affections, 334; *vide* Cerebro-Spinal Fluid

- Lunacy System, Scotch, 780  
 Lymphocytosis of Cerebro-Spinal Fluid, 402
- MAGULA**, Symmetrical Disease of, 241, 242
- Measles, Polyneuritis after, 702
- Medulla: Partial Paralysis of Tongue and Lips due to Anomaly of, 236; Unilateral Congenital Lesion of, 705; *vide* Bulbar Paralysis
- Melancholia: Sensory Disturbances in, 554; Neuraethenic Melancholia, 558
- Meningitis: Basilar, 408, 459; Cerebro-spinal, due to Diplococcus, 311; Hæmorrhagic Fibrinous, 459; Pneumococcal, 107; Lumbar Puncture in, 459; Cytological Examination in, 107; Mercurial Treatment, 459
- Mental condition in Epilepsy, 412
- Mental Diseases: Classification, 662; Metaphysical Conception of, 608; Pre-disposition in, 666; Dispensary Work in, 537; A Non-dementing Psychosis 721; Hysterical Insanity, 611, 723; Neuraethenic Melancholia, 558; Hallucinations of Hearing, 608; Combined Psychoses, 250; Leucocyte Counts in, 666; Cerebro-Spinal Fluid in, 537; Central Nervous System in, 537; Ergotherapy and Psychotherapy, 560; Surgery of Idiocy and Insanity, 670; *vide* also Insanity; Psychoses from Syphilis, 836; Criminal Responsibility, 837; and Heredity, 833
- Metameric Manifestations in Hysteria and Zona, 777
- Methods: of Producing Selective Deposit of Silver in Blocks of Tissue, 522; of obtaining Sections suitable for Toning with Gold, Platinum and other Metals, 522; Selective Staining of Endo-Cellular Fibrillar Reticulum and Peripheral Reticulum of Nerve Cells of Vertebrates, 635; Ramon y Cajal's New Fibril Method, 293, 639; Donath's Method of Demonstrating Choline in Cerebro-Spinal Fluid, 651; Primary Staining of Rat's Brain by Methylene Blue, 689; New Method of Staining Neurofibrils, 146; for Permanent Preservation of Golgi Preparations, 219; Silver-Impregnation of Neurofibrils, 59; Indirect Wallerian Degeneration, 58
- Microcephaly, Brain in, 232
- Middle Finger Phenomenon, 324
- Mind, Relation of Nervous System to, 300
- Morphine Habit; Treatment by Hyoscine, 560
- Multiple Sclerosis, *vide* Disseminated Sclerosis
- Muscles: Paralysis of Involuntary, 148; Paradoxical Contraction, 148; Innervation of Stomach, 442; Orbicular Sphincter in Facial Paralysis, 557; which Move Feathers in Birds, 222; Functional Duality of Muscle, 588; Congenital Absence of Certain, 706
- Muscular Atrophy, Family Neurotic, 703
- Muscular Degeneration, Morphology of, 62
- Muscular Work, Nervous Fatigue and Contraction in, 395; Expenditure of Energy in Dynamic Contraction, 806
- Musculo-Cutaneous Paralysis, 408
- Musculo-Spiral Paralysis, Transplantation of Tendon for, 670
- Myasthenia Gravis, 243, 769, 770, 771, 823
- Myelitis: Acute Transverse, 154, 541; Acute Diffuse, 61; Compression, 704
- Myographic and Ergographic Curve, 298, 590
- Myositis: Polymyositis after Measles, 702
- Myotonic Symptoms in Tetany, 658
- Myxœdema, Abortive, 712
- N-RAYS in Organism, 225
- Necrosis of Maxilla in Tabes, 70
- Nerves: Origin and Intracerebral Course of Motor, 58; Communication between Musculo-Spiral and Ulnar, 221; Union of Fifth Cervical with Superior Cervical Ganglion, 455; Existence of Trophic, 220; of Pulmonary Blood-Vessels, 295; Paralysis of Spinal Accessory, 67; Tumour of Auditory, 77, 603; Motor Function of Vagus, 807; Recurrent Paralysis of Third, 77; Paralysis of Musculo-Cutaneous, Anterior Crural, Supra-Scapular, Sciatic, 408; of Heart in Tabes, 167; Regeneration of, 308, 578, 753; Tendon Transplantation for Musculo-Spiral Paralysis, 670; Surgery of Facial, 417; Stretching of, in Raynaud's Disease, 334; Suture of Fifth and Sixth Cervical in Birth Paralysis of Upper Extremity, 841
- Nerve Cells: Structure of, 56; of Spinal Ganglia, 389; of Sensory Ganglia, 223; Niral Bodies, 56; Dendrites of Cells of Corpora Quadrigemina, 529; Fibrillar Part, 530; Light Pigment in, 389; Paths of Intracellular Conduction, 148; of Ventral Cornua after Section of Posterior Roots, 311; Action of Snake Poison on, 233; Changes in

- Uramic Hemiplegia, 658; Motor Cells at Levels of Origin of Nerves of Extremities, 527; Neurofibrils, 749; *vide* also Methods; Protoplasmic Processes of, 802; Terminal Knobs and Pericellular Net, 802
- Nerve Fibres: Union of Different Kinds, 751; Non-Myelinated, in Foci of Multiple Sclerosis, 304; Positive Coloration in Degeneration, 812; Decussation of Optic Nerve, 807; Bifurcation of, in Spinal Ganglia, 802; Endogenous, 803; Histological Changes in Ununited Divided, 810
- Nerve Roots: Regeneration of Posterior Spinal, 310; Ventral Cornual Cells after Section of Posterior, 311; Distribution of Objective Sensory Disturbances in Acroparæsthesia according to, 323; Degenerations from Lesion of Posterior, 450; Degenerations in Cerebral Tumour, 591; Centrifugal Function of Posterior Roots, 453
- Nerve Tracts: Pyramidal, 56 (2), 377, 439, 440, 698; Cortico-Bulbar and Cortico-Pontine Pyramidal Fibres, 441; Abnormal Bundles in Brain-Stem, 684; Relationship of Abnormal Bundles to Normal Brain Structure, 685; Course and Termination of Gowers' Tract, 685; Intra-Spinal Course of Certain Posterior Roots, 685; Olfactory Radiations, 686; Basal Opticus Root, 386; Tractus Pedunculus Transversus, 386; Intermedio-Lateral, 582; Corpus Restiforme, 804; Bulbo-Cerebellar Connections, 804; Olfactory Tracts, 805
- Nervous Central Organs, Anatomy of, 487
- Nervous System: Anatomy and Physiology of, 164; Relation to Mind, 300
- Neuralgia, Trigeminal, 556; Electric Treatment of Facial, 843
- Neurasthenia: Sensory Disturbances in, 554; Neurasthenic Melancholia, 558; Pathogenesis of Neurasthenic Headache, 607; Isolation and Psychotherapeutics in, 668
- Neuritis: Optic, in Intracranial Tumour, 589; Diabetic, 645; Polyneuritis after Measles, 702; Neuritis Periaxilis, 646
- Neurofibrils, 749
- Neurofibromatosis, 702
- Neuroglial Changes in Intracranial Tumour, 589
- Neuromata, Acoustic, 603
- Neuromyelitis, Acute Optic, 314
- Neurone Theory, 146, 402, 640, 690, 691
- Neuronophagia, 220
- Neuroses, Variation in Relation to Origin of, 285
- Neurotic muscular atrophy, 703
- Nissl's Bodies, 56
- Noises in Head, 759
- Nucleus Lateral to the Fasciculus Solitarius, 55
- OBITUARY Notice: Friedrich Jolly, 169; Karl Weigert, 671
- Obsessions and Impulsions, 85
- Obstetrical Paralysis, 757
- Occipital Lobe, Tumours of, 74
- Ocular Movements: Cerebral Connections of, 392; Conjugate Deviation of head and eyes with Homonymous Hemianopia, 472; 807; Conjugate Deviation of, 660
- Oculo-motor Nerve: Recurrent Paralysis, 77; Paralysis of, in Tabes Dorsalis, 647
- Oedema: Hemiodema in Hemiplegia, 777; Trophœdema, 778
- Olfactory Radiations, 686
- Optic Nerve: Course of Fibres, 386; Reflex Apparatus, 748; in Tabetic Amaurosis, 813
- Optic Neuritis in Intracranial Tumour, 589; Pathology and Pathogeny of Choked Disc, 643
- PAIN in Brain Tumours, Relief of, 88
- Pains, Recurring, 244
- Palate, Paralysis of, 777
- Palpebral Symptoms in Facial Paralysis, 163, 322
- Paradoxical Contraction, 148
- Paralysis, Obstetrical, 757
- Paralysis Agitans, 462, 465, 822; Exercise Treatment of, 613
- Paramnesia, 226
- Paraplegia: Mode of Turning in Walking in Spastic, 748; Family Spastic, 762
- Parkinson's Disease, *vide* Paralysis Agitans
- Partial Paralysis of Upper Limb from Vascular Lesion of Cord, 69
- Pedunculus Corporis Mamillaris, 385
- Peripheral Paralysis, *vide* Nerves
- Phosphoric Acid in Cerebro-Spinal Fluid, 652
- Photisms, Stability and Hereditary Transmission of, 302
- Plantar Reflex, *vide* Reflexes
- Platinum Method, *vide* Methods
- Pneumococcus Lanceolatus, Post-Basal Meningitis due to, 408
- Pons: Structure of, 687; Solitary Tubercle of, 601; Hemorrhage, 705
- Porencephalic Brain, 406



Posterior Columns, Anatomy of, 391  
 Posterior Roots, Centrifugal Function of, 453  
 Potts' Disease, Mid-dorsal, 704  
 Progressive Paralysis, *vide* General Paralysis  
 Pseud-Affective Reflex, Spinal Path of a New, 695  
 Pseudo-Tetany, 772  
 Psychiatry, Comparative, 668; Importance of Lumbar Puncture for, 479  
 Psychology of Joan of Arc, 582  
 Psycho-Epilepsy of Janet, 612  
 Psychopathic Wards, etc., in America, 780  
 Psychopathology of Everyday Life, 489  
 Psychoses, Combined, 250  
 Psychotherapy and Ergotherapy, 560  
 Psychotherapeutics in Neurasthenia and Hysteria, 468  
 Pulmonary Blood-Vessels, Innervation of, 295  
 Pupil: "Lidschlussreaction" of, 76; Measurement of, 257; Light Reaction of, 162, 257; Dilatation Reflex, 393; Prolongation of Reaction of, 318; Hemianopic Immobility, 161; Movement of Eyeball accompanying Light Reaction, 162; Vertical Eye-Movement Testing in Positions of Abduction and Adduction, 799; Light Reflex, 807; Inequality in Diseases of Lungs and Pleura, 829  
 Pyramidal Tracts, *vide* Nerve Tracts  
 Pyridin, Action upon Nervous Tissues, 685  
 RADIUM, Action on Cerebral Cortex, 896  
 Radio-Active Bodies and Insanity, 781  
 Raynaud's Disease: Stretching of Nerves in, 334; Its Relation to Erythromelalgia, 468  
 Recurrent Paralysis of Third Nerve, 77  
 Recurring Pains, 244  
 Reflexes: Influence of Fatigue on, 297; Superposition of, 555; Facial, 247, 474; Pupil, 393; New-pino-Muscular Phenomenon, 164; Pseud-Affective, and its Spinal Path, 695; Optic Reflex Apparatus, 748; Corneo-mandibular, 163; Superficial, 248, 296; Plantar, 248, 415, 345; Babinski's Sign in Extra-Pyramidal Lesions, 825; Cremasteric, 555; Gluteal, 714; Tendon Jerks, 554; Knee-Jerk, 587; Ankle Clonus, 556; Triceps, Biceps, and Finger Clonus, 85; Reflex on Dorsum of Foot, 416; Flexor Reflex of Toes, 714; Movements in Infantile Spastic Diplegia, 84; Excitability of Upper

Limb, 324; Phenomena in Extremities in Central Organic Paralysis, 473  
 Regeneration: of Nerve, 308, 310, 578, 753, 810; in Spinal Cord, 587  
 Retina: Finer Structure of, 683; in Tabetic Amaurosis, 755; Changes in Macula Region, 241, 242; Projection on Calcarine Cortex, 75  
 Respiratory Movements: of Glottis, 394; in Hemiplegia, 161; of Superior Inter-costal Muscles in Hemiplegia, 160; of Thorax and Diaphragm, 150  
 Retro-Calcarine Region of Cortex Cerebri, 442  
 Reviews: Allgemeine Anatomie und Physiologie des Nervensystems, 164; Les Nerfs du Cœur chez les Tabétiques, 187; Fatigue, 255; Journal de Psychologie Normale et Pathologique, 256; Die Messung der Pupillengrösse und Zeitbestimmung der Lichtreaction der Pupillen, 257; Arbeiten aus dem Neurologischen Institute an der Wiener Universität, 336; Vorlesungen über den Bau der nervösen Zentral-Organen des Menschen und der Thiere, 487; Psycho-pathology of Everyday Life, 489; Des Scléroses Combinées de la Moelle, 616; Cécité et Tabes, 721; Hysterical Mental Affections, 723; Nervous Affections of the Heart, 783; Heredity and Genetic Variation, 847  
 Ribs, Cervical, 830  
 Rolandic Region, Tumours of, 818  
 Röntgen Rays in Neurology, 608  
 Root Areas, Demarcation of, by Tumour of Cauda Equina, 238; Cutaneous Segmentation Postaxial to Lower Limb, 816  
 Roots, Nerve, *vide* Nerve Roots  
 SCAPULA: Congenital High Position of, 558  
 Sciatic, Paralysis of, 409  
 Sciatica, with Kypho-Scoliosis, 549; Oscillating Current in, 844  
 Sclerosis, Combined, 431, 616, 648; Combined Tabetic, 458; Primary of Posterior Columns, 97; *vide* also Disseminated Sclerosis  
 Segmentation of Intermedio-Lateral Tract, 583  
 Segmentation Postaxial to Lower Limb, 816  
 Semi-Circular Canal: Involuntary Movements of Head as Expression of Isolated Affection of, 324; Removal in Aural Vertigo, 669  
 Sensation: Visceral Anaesthesia in General Paralysis, 470; in Acroparæsthesia,

- 823; Pathway for Taste, 601; Pain in Cerebral Hemiplegia, 775
- Sensory Root Areas of Lower Limbs, Demarcation of, by Tumour of Cauda Equina, 233
- Sero-Therapy of Epilepsy, 482, 782; of Exophthalmic Goitre, 782; Antiserum in Acute Insanity, 483
- Silver Method, *vide* Methods
- Sleep, Pathological, in Cerebral Tumour, 763
- Sleeping Sickness: Tissues in, 593
- Snake Poison, Action on Nervous System, 233, 809
- Sodium, Acid Phosphate of, in Alkalinity of Urine, 88
- Solar Syndromes, Experimental, 534
- Speech, Cerebral Zone of, 826; *vide* Aphasia
- Sphincters, Innervation of, 642
- Spinal Accessory Nerve, Paralysis of, 67
- Spinal Cord: *Anatomy*: Structure of Posterior Columns, 143, 391; of Pons, 449; Intracellular Network of Golgi in Cells of, 143; Endogenous Fibres, 803. *Physiology*: Centrifugal Function of Posterior Roots, 453. *Pathology*: Changes in Tadpoles, after Amputation of Limbs, 751; Secondary Degeneration in Anterior Column, 693; Degenerations from Transverse Lesion of, 450; Regeneration in, 587; Compensatory Processes in, 699; Radicular Lesions in Cerebral Tumour, 591. *Clinical*: Total Transverse Lesion of, 700; Subacute Combined Degenerations, 97; Secondary Carcinoma of, 154; Partial Paralysis of Upper Limb from Vascular Lesions of Cord, 69; Senile Combined Sclerosis, 431; Syringal Hemorrhage, 543; Multiple Sclerosis of, 546; Unilateral Lesion of, 547; Congenital Defect of, 705; Cirroid Aneurism of Cervical Cord, 545; Intra-Medullary Abscess of, 819; Injury to Lower Cord and Cauda Equina, 819; *vide* also Disseminated Sclerosis, Tabes, Tumours, Myelitis, etc.
- Spinal Deformities: Sciatica with Kyphoscoliosis, 549; Rheumatic Spondylosis and Ankylosis, 550; Kyphosis of Articular or Muscular Origin, 550; in Tabes, 758.
- Spinal Muscular Atrophy, Progressive, in Early Childhood, 315
- Sprengel's Shoulder, 558
- Status Epilepticus, 767
- Stereognostic Sense, *vide* Astereognosis
- Stomach: Muscular Innervation of, 642; Tetany in Dilatation of, 655; Tetany in Diseases of, 656
- Subacute Combined Sclerosis, *vide* Combined Sclerosis
- Superposition of Reflexes, 555
- Suprarenal Extract, Action of, 295
- Suprascapula Paralysis, 409
- Sympathetic System of Birds, 222
- Syndrome of Benedict, Inferior, 156
- Syphilis: of Nervous System, 764; and Progressive Paralysis, 836; Psychoses from, 836
- Syringobulbia and Syringomyelia, 704
- Syringomyelia: Leprosy or, 650; with Cheiromegalia, 69; Syringal Hemorrhage into Cord, 543; Pathological Histology and Pathogeny of, 815; and Syringobulbia, 704
- TABES Dorsalis: Nature of, 307; Spinal Root Ganglia in, 697; Optic Nerve in, 813; Nerves of Heart in, 167; Vertebral Disease in, 758; Spinal Accessory Paralysis, 67; Retina in Tabetic Anurosis, 755; Paralysis of Oculo-motor Nerves, 647; Buccal Perforation in, 70; Multiple Necrosis of Maxilla in, 70; Unusual Cases, 177; and Tabo-paralysis in Childhood and Youth, 539; Hereditary Syphilitic, 445; Cytodiagnosis in, 540; Influence of Fever on Pains of, 538; Diagnosis and Treatment, 643; Optic Nerve in Tabetic Amaurosis, 813; Tabetic Gastric Crisis, 817; and General Paralysis, 71
- Temporo-sphenoidal Lobe, Tumour of, 74
- Tendon Jerks, *vide* Reflexes
- Tendon Transplantation for Musculo-spiral Paralysis, 670
- Tetanus, Cephalic, with Facial Diplegia, 320
- Tetany: Gastric, 159; "Atypical," 654; in Dilatation of Stomach, 655; in Diseases of Stomach, 656; Thyroid Gland and, 657; with Myotonic Symptoms, 658; True and Hysterical, 772
- Third Nerve, Recurrent Paralysis of, 77
- Thorax and Diaphragm, Respiratory Movements of, 150
- Thyroid Extract in Puerperal Eclampsia, 835
- Thyroid Gland and Tetany, 657
- Tic of the Eyes, 238; Bilateral Spasm of Muscles of Neck and Face, 825; of Childhood, 824
- Toes, Flexor Reflex of, 714
- Tongue: Atrophy of, in Erb's Disease, 771
- "Transitory Fissures" of Brain, 442
- Trapezius, Innervation of, 707
- Trauma in Relation to Disease of Nervous System, 593

**Tremors**, 776  
**Triceps**, Biceps, and Finger Clonus, 85  
**Trigeminal Neuralgia**, 556  
**Trophœdema**, 778  
**Trophic Nerves**, Existence of, 220  
**Tuberculosis of Nervous System**, 64  
**Tumour**: *Brain*, 462; of Rolandic Region, 318; of Occipital Lobe, 74; of Temporo-sphenoidal Lobe, 74; of Postero-Parietal Region, 156; of Ponto-medullo-cerebellar Space, 603; of Centrum Ovale, 709; Meningeal, 487; Congenital Cysts of Fourth Ventricle, 592; Psammomasaroma, 602; Optic Neuritis in, 569; with Clinical Appearance of G. P., 551; Pathological Sleep in, 783; Radicular Lesions of Cord which accompany, 591; Method of Relief of Pain in, 88; Ligature of Vessels in Meningeal Tumour, 487; Hemiplegia as Sign of, 824. *Spinal Cord*: 89; Upper Cervical Cord and Medulla Oblongata, 707; Intradural of Cervical Meninges, 595; Can it Diminish Spontaneously in Size? 68  
 Of Cauda Equina, 233  
 Of Auditory Nerve, 77, 603

**UMBILICUS**, Sensory Segmental Zone of, 681

**Uncinate Group of Fits**, 552

**Uremic Hemiplegia**, 658

**VAGUS**: Motor Function of, 807  
 Variation and Heredity, Morphological Continuity of Germ Cells as Basis of, 1, 114, 185  
**Vertebral Disease**: in Tabes, 758; *vide* also Spinal Deformities  
**Vertigo**, Lumbar Puncture in, 319; Removal of Semi-circular Canals in Aural, 669  
**Vessels**, Ligature of, in Meningeal Tumour, 487  
**Vibratory Sensibility**, 774  
**Visceral Anæsthesia**, 470  
**Vision**: Projection of Retina on Calcarine Cortex, 75; Binocular and Stereoscopic, 807; Cortical Localisation of, 820  
**Voice** in General Paralysis, 72  
**Vomiting**, Fæcal and Reversed Peristalsis in Functional Nervous Disease, 830

**WEICHSELBAUM**, Cerebro-Spinal Meningitis due to Diplococcus of, 311

**Weigert**, Professor Karl, Obituary Notice, 671

**Whooping-Cough**, Nervous Complications of, 772

**Word Blindness**, *vide* Alexia

**ZONA**, Metamerie Manifestations in, 777

## AUTHORS' INDEX.

**ABADIE**, Jean, and Henri Verger. Plan-  
 tar Reflex, 415  
**Adamkiewicz**, A. True Motor Centres,  
 584  
**Alaux** and Delucq. Adiposis Dolorosa,  
 771  
**Alessi**. Pathological Anatomy of Acute  
 Delirium, 458  
**Alter**, W. Certain Symptoms in Cata-  
 tonic States, 329  
**Alzheimer**. Delirium Alcoholicum Feb-  
 rile of Magnan, 662; Anatomical Basis  
 of Idiocy, 698  
**Anderson**, H. K. Paralysis of Involun-  
 tary Muscle, 148  
**Anderson**, H. K. and J. N. Langley.  
 Union of Fifth Cervical Nerve with  
 Superior Cervical Ganglion, 455;  
 Union of Different Kinds of Nerve  
 Fibres, 751; Autogenetic Regeneration  
 in Nerves of Limbs, 758  
**Angiollola** and Galdi. Psychoses from  
 Syphilis, 836

**Apert**, E. Abortive Myxœdema, 712  
**Ascherson**, W. L. Tumour of Centrum  
 Ovale, 709  
**Aufschlager**. Hemichorea, 286

**BABINSKI**. Cutaneous Reflexes in Affec-  
 tions of Pyramidal Tract, 296; Treat-  
 ment of Auricular Vertigo by Lumbar  
 Puncture, 334; Hemorrhagic Fibrin-  
 ous Meningitis, etc., 459  
**Ballet** and Laignel-Lavastine. Acute  
 Myelitis in Secondary Carcinoma of  
 Dura Mater or Cord, 154  
**Barnes**, Stanley. Diagnostic Value of  
 the Plantar Reflex, 345  
**Bartels**, M. Trigeminal Neuralgia and  
 Recurrent Corneal Erosion, 556  
**Batten**, F. E. Cerebral Degenerations  
 with Symmetrical Changes in Macula  
 in Two Members of a Family, 242  
**Batten**, Rayner D. Two Brothers with  
 Symmetrical Disease of Macula, 241  
**Bauer** and Brissaud. Spinal Cord of

- Tadpole Consecutive to Amputation of Limbs, 751
- Beyon. Central Nervous System in Cretins, 457
- Beard, J. Morphological Continuity of Germ-Cells as Basis of Heredity and Variation, 1, 114, 185
- Bechterew, von. Reflex Phenomenon in Extremities in Central Organic Paralysis, 473; Musculus Orbicularis Oculi in Facial Paralysis, 557; Gluteal Reflex, 714
- Beever, Charles, and James Collier. Cortical Localisation of Vision, 820
- Belmondo, E. Myographic and Ergographic Curve, 298, 590
- Berger, E. and E. Loewy. Secondary Contracture of Levator Palpebræ Superioris in Facial Paralysis, 163
- Bernhardt. Rare Peripheral Paralyzes, 408; Neuritis Periaxialis, 646
- Bernheimer. Cerebral Connections of Ocular Movements, 392
- Berry, Walter D. Intracranial Psammoma without Paralysis, 602
- Bertolotti. Babinski's Sign in Extra-Pyramidal Lesions, 825
- Bethe, A. Anatomy and Physiology of the Nervous System (Review), 164; Neurone Theory, 690
- Beule, Fritz de. Respiratory Movements of Glottis in Rabbit, 394
- Bianchini, M. I. Ergotherapy and Psychotherapy, 560
- Bielschowsky, Max. Silver Impregnation of Neurofibrils, 59; Non-Myelinated Nerve Fibres in Foci of Multiple Sclerosis, 304
- Bielschowsky and Pollack. Innervation of Mammalian Eye, 528
- Bignami and Marchiafava. Corpus Callosum in Alcoholism, 153
- Bikeles, G. Arrangement of Motor Cells, 527; Regeneration in Spinal Cord, 587
- Bing, Robert. Endogenous Spinal Fibres, 803
- Blumer, Hun and Streater. Myasthenia Gravis, 769
- Bochenheimer. Facial Nerve in Relation to Surgery, 417
- Boidon and Chauffard. A Year's Lumbar Punctures, 599; Lumbar Punctures, 650
- Bonhoeffer. Korsakoff's Symptom Complex in Relation to Various Diseases, 610
- Bonnamour, S. and A. Pic. Friedreich's Disease with Cerebral Softening, 541
- Bonnefoy. Therapeutic Action of Currents of High Frequency and High Tension, 484
- Boston and Pearce. Blood in Epilepsy, 535
- Bouchaud. Cephalic Tetanus and Facial Diplegia, 520; Infantile Cerebral Hemiplegia and Hemiataxia, 824
- Boutenko and Soukhanoff. Korsakoff's Disease, 245
- Box, C. R. Tumour of Cauda Equina, 233
- Bradley, O. Charnock. Neuromeres of the Rhombencephalon of the Pig, 635; Mammalian Cerebellum, 744
- Bramwell, Byrom. Disseminated Sclerosis, 462; Unilateral Lesion of Spinal Cord; Brown-Séquard's Paralysis, 547
- Brausch, Martin. Family Neurotic Muscular Atrophy, 703
- Brécy and Brissaud. Acute Optic Neuromyelitis, 314
- Breuer and Marburg. Apoplectiform Bulbar Paralysis, 316
- Brissaud, E. Recurring Pains, 244
- Brissaud and Bauer. Spinal Cord of Tadpole Consecutive to Amputation of Limbs, 751
- Brissaud and Brécy. Acute Optic Neuromyelitis, 314
- Brissaud and H. Grenet. Kyphosis of Articular or Muscular Origin, 550
- Brissaud and Meige. Infantile Type of Giantism, 713
- Broc and Merklen. Paralysis of Palate, 777
- Brodie, T. G., and W. E. Dixon. Innervation of Pulmonary Blood-vessels, 295
- Browning, William. Method for Relief of Pain in Brain Tumours, 88
- Bruce, Alexander, and C. Macfie Campbell. Segmentation of the Intermedialateral Tract, 583
- Bruce, Alexander, and W. B. Drummond. Intra-Cranial Aneurism, 737
- Bruce, Lewis C. Anti-serums in Acute Insanity, 483
- Bruce, Lewis C., and A. M. S. Peebles. Leucocyte Counts in Mental Disease, 666
- Bruns, L. Hysteria in Childhood, 157; Paralysis Agitans, 822
- Brush, A. C. Meningeal Tumour treated by Ligature of the Vessels, 487
- Buchanan, J. M. Treatment of Morphine Habit by Hyoscine, 560
- Buck, D. de. Psycho-Epilepsy of Janet, 612
- Buck, D. de, and L. de Moor. Morphology of Muscular Degeneration, 62
- Bullard, W. N. Astereognosis in Cerebral Affections, 470
- Bunzl, Victor. Parasitology of Brain, 701

- Burr, C. W. Fever in Locomotor Ataxia, 538
- Bury, Judson S. Respiratory Movements in Hemiplegia, 161; Trauma in Relation to Disease of Nervous System, 593
- Buzzard, E. Farquhar, and James Collier. Degenerations from Lesions of Posterior Nerve Roots and from Transverse Lesions of Cord, 450
- Buzzard, T. Remissions and Relapses in Insular Sclerosis, 818
- Bychowski, Z. Hypotonus and Hyper-tonus, 775
- CABOT, Richard. Alcohol and Arterio-sclerosis, 833
- Cajal, Ramon y. New Method for Staining Neuro-Fibrillae, 146, 293
- Camp and Spiller. Multiple Sclerosis, 649
- Campbell, C. Macfie. Lumbar Puncture, 39
- Campbell, C. Macfie, and Alexander Bruce. Segmentation of the Inter-medio-Lateral Tract, 533
- Cestan, R., and Dupuy-Dutemps. Constant Palpebral Symptom in Peripheral Facial Palsy, 322
- Cestan and Raymond. Cirroid Aneurism of Cervical Cord, 545
- Charpentier, Augustin. The N-Rays in the Organism, 225
- Chauffard and Boidon. A Year's Lumbar Punctures, 599; Lumbar Punctures, 650
- Chauveau, A. Muscular Work in Dynamic Contraction, 806
- Chavigny. Metamerie Manifestations in Zona and Hysteria, 777
- Chenzinski, C. Structure of Nerve Cells (What are Nissl's Bodies?), 56
- Chompret, J. Buccal Perforation or Multiple Necrosis of Maxilla in a Tabetic, 70
- Clark, L. Pierce. Movements of Muscles in Hemiplegia, 160
- Clark, L. Pierce, and Alan Montgomery. Psychopathic Wards, Pavilions and Hospitals for American Cities, 780
- Clark, L. Pierce, and T. P. Prout. Status Epilepticus, 767
- Coester. Diagnosis and Treatment of Tabes, 648
- Collier, James. Effects of Total Transverse Lesion of Spinal Cord, 700
- Collier, James, and E. Farquhar Buzzard. Degenerations from Lesions of Posterior Nerve Roots and from Transverse Lesions of Cord, 450
- Collier, James, and Aldren Turner. In-tra-Medullary Abscess of Spinal Cord, 819
- Collier, James, and Charles Beevor. Cortical Localisation of Vision, 820
- Perineal Zoster, 816
- Collins, Joseph. Primary Sclerosis of Posterior Columns, 97
- Combe. Inferior Syndrome of Benedict, 156
- Copp, Owen. Scotch Lunacy System, 780
- Coriat, J. H. Cerebro-Spinal Fluid and Central Nervous System in Mental Diseases, 537
- Cornu, E. Cerebral Tumour with Clinical Appearance of General Paralysis, 551
- Crouzon, O. Tabetic Combined Sclerosis, 458; Des Sclérosis Combinée de la Moelle (Review), 616
- Crouzon and Loeper. Hemiedema in Hemiplegia, 777
- Crouzon, O., and Kinnier Wilson. Senile Combined Sclerosis of Spinal Cord, 431
- Curschmann, H. Pseudotetany, 772
- Cushing, Harvey. Intracranial Tumour of Cervical Meninges, 595; Perineal Zoster, 816
- Czarniecki. Dendrites of Nerve Cells of Anterior and Posterior Corpora Quad-rigemina, 529
- DA COSTA. Surgery of Idiocy and Insanity, 670
- Dana, Charles L. Problem of Well-to-do Inebriate, 85; Cytodiagnosis in Nervous Diseases, 401; Facial Responses and Reflexes, 474; A Non-Dementing Psychosis, 721
- Dana, Charles L., and Joseph Fraenkel. Aphasia with Sensory Anomia, 606
- Dean, George, and C. H. Usher. Course of Optic Nerve Fibres, 836
- De Bovis. Stretching of Nerves in Raynaud's Disease, 334
- Debray. Neurone Theory, 402
- Decroly. Dysarthritic Trouble in Epileptic Child, 237
- Dehérain. Inequality of Pupils in Lung Diseases, 829
- Dejerine, M. and Mme. Direct Pyramidal Tract, 439
- Déjérine and Egger. Objective Sensory Disturbances in Acroparesthesia, 323
- Déjérine and Thomas. Verbal Blindness with Agraphia, 658; Sensory Aphasia, 711
- Delherm and Laquerrière. Mechanism of Reaction of Degeneration, 832
- Delucq and Alanx. Adiposis Dolorosa, 771

- Delupis.** Leprosy or Syringomyelia, 650
- Dercurm, F. X. and W. W. Keen.** Tumour of Postero-Parietal Region, 156
- Diefendorf.** Blood Changes in Dementia Paralytica, 154
- Dinkler.** Acute Transverse Myelitis, 541; Multiple Sclerosis of Brain and Spinal Cord, 546
- Dixon, W. E. and T. G. Brodie.** Innervation of Pulmonary Blood-vessels, 295
- Donaggio, Arturo.** Relationship between Paths of Inter-cellular Conduction and Periphery of Nerve Cell, 148; Action of Pyridinupon Nervous Tissues, 685; Positive Coloration of Nerve Fibres in Primary and Secondary Degeneration, 812
- Donath.** Cholin in Cerebro-Spinal Fluid in Epilepsy and Organic Nervous Diseases, 62; Phosphoric Acid in Cerebro-Spinal Fluid in General and in Nervous Diseases, 652
- Doumer.** Electrotherapy in Nervous Disease, 842
- Drummond, W. B., and Alexander Bruce.** Intra-Cranial Aneurism, 737
- Dubois, M.** Sensory Disturbances in Neurasthenic and Melancholic States, 554
- Dufour, H.** Conjugate Deviation of Head and Eyes, with Homonymous Hemianopsia, 472
- Dumez, Jules.** Psychology of Joan of Arc, 532
- Dunton, W. R.** Dementia Præcox, 610
- Dupuy-Dutemps and R. Cestan.** Constant Palpebral Symptom in Peripheral Facial Palsy, 322
- Durante, G.** Neurone Theory, 146, 640
- Duret, H.** Tumours of Occipital Lobe and of Temporo-Sphenoidal Lobe, 74; Tumours of Rolandic Area, 318
- EDENS and Jessen.** Polymyositis and Polyneuritis after Measles, 702
- Edinger.** Anatomy of the Nervous Central Organs (Review), 487
- Edmunda, Arthur, F. W. Mott, and W. D. Halliburton.** Regeneration of Nerves, 586
- Egger and Déjérine.** Objective Sensory Disturbances in Acroparesthesia, 323
- Elliott, J. W. and J. J. Putnam.** Tumour involving Spinal Cord treated by Operation, 89
- Eshner, Augustus.** Ankle Clonus, 556
- Esposito, G.** Neuronophagia, 220
- Evensen, Hans.** Chronic Alcoholism, 325
- FEINDEL and Meige.** Tics of Childhood, 824
- Fenn, S. Blackwell.** "Gold Cure" of Inebriety, 561
- Féré, C.** Hypotonic Flat Foot in General Paralysis, 414
- Ferguson, Hinschelwood and Macphail.** Word-Blindness, with Right Homonymous Hemianopsia, 827
- Fischer.** Progressive Facial Hemiatrophy, 79
- Flashman, J. Froude.** Parieto-Occipital Fissure, 217; Sulci of Brains of Australian Aborigines, 218; Brain of Microcephalic Idiot, 232
- Fleming, R. A.** Optic Neuritis in Intracranial Tumour, 569
- Foerster, Richard.** Alexia in Achondroplasia and in an Imbecile, 411
- Forestier, H.** Sciatica with Kyphoscoliosis, 549; Rheumatic Spondylitis and Ankylosis, 550
- Fraenkel, J.** Lymphocytosis of Cerebro-spinal Fluid, 402
- Fraenkel, Joseph, and C. L. Dana.** Aphasia and Sensory Anomia, 606
- Fraenkel, Joseph, and Ramsay Hunt.** Neuro-fibromatosis, 702; Tumours of Ponto-medullo-cerebellar Space, Acoustic Neuromata, 603
- Franchesi.** Pathological Sleep in Cerebral Tumour, 763
- Frank.** Vertebral Disease in Tabes Dorsalis, 758
- Frazier, Charles H. and W. G. Spiller.** Regeneration of Posterior Spinal Roots, 310
- Frenkel, H. S.** Cytodiagnosis in Tabes and General Paralysis, 540
- Freud, S.** Psychopathology of Everyday Life (Review), 489
- Freund.** Movement of Eyeball accompanying Pupillary Light Reaction, 162
- Friedlaender, R.** Exercise Treatment in Paralysis Agitans, 618
- Friedländer and Kempner.** Hemianopic Immobility of Pupil, 161
- Friedmann.** Neurasthenic Melancholia, 558
- Frohlich, A.** Intra-Spinal Course of Certain Posterior Roots, 685
- Fuchs.** A Facial Reflex, 247; Messung der Pupillengrösse (Review), 257; Idiopathic Chronic Internal Hydrocephalus, 759
- Funke, W.** Cerebro-Spinal Fluid of Syphilitics, 540
- GALDI and Angiolella.** Psychooses from Syphilis, 886
- Gallavardin and Weill.** Pathological

- Anatomy of Acute Diffuse Myelitis, 16
- Gaupp, Robert. Combined Psychoses, 251
- Gaussen. Bilateral Spasm of Muscles of Neck and Face, 825
- Geluchten, A. van. Researches on Origin and Intracerebral Course of Motor Nerves by Means of Indirect Wallerian Degeneration, 58; Central Connections of Deiters' Nucleus and its Neighbouring Nuclei, 383; Terminal Knobs and Pericellular Net, 802; Corpus Restiforme and Bulbo-cerebellar Connections, 804; Olfactory Tracts, 805
- Geier, Soukhanoff, and Gourévitch. Protoplasmic Processes of Nerve-Cells, 802
- Gentile, E. Influence of Electricity on Cerebral Pulse, 591
- Gerhartz. Blood-Serum Treatment of Epilepsy, 782
- Gibson, G. A. Nervous Affections of the Heart (Review), 783
- Goldflam, S. Aetiology and Symptomatology of Intermittent Claudication, 81; Skin Reflexes of Lower Extremities, 248
- Goldstein, Kurt. Posterior Columns of Spinal Cord, 143
- Gourévitch, Soukhanoff, and Geier. Protoplasmic Processes of Nerve-Cells, 802
- Gowers, Sir W. R. Syringal Hemorrhage, 548; Insanity and Epilepsy in Relation to Life Assurance, 778
- Grasset, J. Conjugate Deviation of Eyes and Hemianopia, 660
- Gray, H. W. M. Transplantation of Tendon for Musculo-Spiral Paralysis, 670
- Grenet, H., and E. Brissaud. Kyphosis of Articular or Muscular Origin, 550
- Guidi. Auto-Intoxication in Epilepsy, 319
- Guillain, Georges, and Pierre Marie. Direct Pyramidal Tract and "Oreocent" Pyramidal Tract, 56; Homolateral Pyramidal Tract, 56; Secondary Degenerations of Anterior Column of Cord, 693
- Guilleminot, H. The N-Rays, 225
- Gulbenk. Dysantigraphia, 322
- HAGELSTAM, Jarl. Tabes and Tabo-Paralysis in Childhood and Youth, 539
- Hall and Walbrach. Adipositas Dolorosa, 713
- Halliburton, W. D., F. W. Mott, and Arthur Edmunda. Regeneration of Nerves, 586
- Hamilton, Arthur S. Erythromelalgia, 536
- Hanke, Victor. Brain in Congenital Bilateral Anophthalmus, 456
- Hannay, Mary Baird. Porencephalic Brain, 406
- Harris, Wilfred. Post-Diphtheritic Bulbar Paralysis, 461, 652; Brachial Plexus and its Motor Distribution, 688; Binocular and Stereoscopic Vision, 807
- Hart, T. S. Paralysis Agitans, 464
- Hatschek. Pedunculus Corporis Mammillaris, Ganglion Tegmenti Profundum, and Dorso-Ventral Raphe-Fibres of Tegmentum, 385; Ventral Tegmentum Region, Median Fillet and Structure of Pons, 687
- Hauser and Thomas. Changes in Spinal Root Ganglia of Tabetics, 697; Pathological Histology and Pathogeny of Syringomyelia, 815
- Heitz, Jean. Nerves of Heart in Tabes (Review), 167
- Henneberg. Ganser's Symptom, 607
- Henriksen, Paul B.. Nerve Suture and Nerve Regeneration, 308
- Henschen, S. E. Can Spinal Cord Tumour Diminish in Size Spontaneously? 68; Projection of Retina on Calcarine Cortex, 75
- Hejburn and Waterston. Spinal Cord of Porpoise, 449
- Hibbs, Russell, A., and H. Correll-Loewenstein. Congenital High Position of Scapula, 558
- Hinshelwood, James. Congenital Word-Blindness; 829
- Hinshelwood, Macphail, and Ferguson. Word-Blindness, with Right Homonymous Hemianopia, 827
- Hoffmann. Progressive Facial Hemihypertrophy, 80
- Honigmann. Gastric Cancer, and Tetany in Diseases of Stomach, 656
- Hun, Blumer, and Streater. Myasthenia Gravis, 769
- Hunt, J. Ramsay. Congenital Cysts of the Fourth Ventricle, 592; Multiple Sclerosis with Dementia, 598; Course and Termination of Gowers' Tract, 685; Mid-dorsal Potts' Disease, Compression-Myelitis, 704; Juvenile Apoplexy, 708
- Hunt, J. Ramsay, and Joseph Fraenkel. Tumours of the Ponto-medullo-cerebellar Space, Acoustic Neuromata, 603; Neurofibromatosis, 702
- Hunter, W. K., and George Lamb. Action of Snake Poison on Nervous System, 233; Action of Venoms of Snakes on Nervous System, 809
- Hutchison, Robert. Acid Phosphate of Sodium in Alkalinity of Urine, 88

- IRKLSOHN.** *Etiology of Intermittent Claudication*, 81
- JACOBI, Josef.** *Thyroid Gland and Tetany*, 657
- Jelliffe, S. E.** *Dispensary Work in Nervous and Mental Diseases*, 537; *Multiple Sclerosis*, 597
- Jessen and Edens.** *Polymyositis and Polyneuritis after Measles*, 702
- Joffroy and Raband.** *Association of Tabes with General Paralysis of Insane*, 71
- Jones, Robert.** *Relation of Inebriety to Insanity*, 716; *Radio-active Bodies and Insanity*, 781
- Jotzko.** *Functional Duality of Muscle*, 588
- KAMPFERSTEIN.** *Pathology and Pathogenesis of Choked Disc*, 648
- Kaplan, J.** *Corneomandibular Reflex*, 163
- Karplus, J. P.** *Grey Matter of Funiculus Cuneatus of Human Medulla Oblongata*, 687
- Karplus, J. P., and A. Spitzer.** *Abnormal Bundles in Human Brain-Stem*, 684
- Keen, W. W., and F. X. Dercum.** *Tumour of Postero-Parietal Region*, 156
- Kennedy, Robert.** *Histological Changes in Ununited Nerves*, 810; *Treatment of Birth Paralysis of Upper Extremity*, 841
- Kempner and Friedländer.** *Hemianopic Immobility of Pupil*, 161
- Kidd, Leonard J.** *Mode of Turning, in Organic Hemiplegia and Spastic Paraplegia*, 742; *Vertical Eye-Movement Testing*, 799; *Tabetic Gastric Crisis*, 817
- Knapp and Thomas.** *Reflexes in Long Distance Runners*, 297
- Koenig, W.** *Etiology of Simple Idiocy*, 475
- Kohlbrugge, J. H. F.** *Cerebral Fissure in Apes*, 442
- Kohn, Rudolph.** *Pseudo-Paralytic Myasthenia Gravis and Pregnancy*, 243
- König, W.** *Heredity from Psychiatric Aspect*, 833
- Kraepelin.** *Comparative Psychiatry*, 668
- Kreuzfuchs, Siegmund.** *Dilatation Reflex of Pupil to Diminished Illumination*, 393
- Kronthal.** *Relation of Nervous System to Mind*, 800
- LADAME, Charles.** *Nervous System in Experimental Hydrophobia*, 405
- Laignel-Lavastine.** *Experimental Solar Syndromes*, 534
- Laignel-Lavastine and Ballet.** *Acute Myelitis in Secondary Carcinoma of Dura Mater or Cord*, 154
- Lake, Richard.** *Removal of Semi-circular Canals in Unilateral Aural Vertigo*, 669
- Lamb, George, and W. K. Hunter.** *Action of Snake Poison on Nervous System*, 233; *Action of Venoms of Snakes on Nervous System*, 809
- Langley, J. N.** *Sympathetic System of Birds*, 222
- Langley, J. N., and H. K. Anderson.** *Union of Fifth Cervical Nerve with Superior Cervical Ganglion*, 455; *Union of Different Kinds of Nerve Fibres*, 751; *Auto-Genetic Regeneration in Nerves of Limbs*, 753
- Laquerrière and Delherm.** *Mechanism of Reaction of Degeneration*, 832
- Latzko and Steinberg.** *Anencephaly*, 152
- Lemaitre, Aug.** *Paramnesia*, 226; *Coloured Audition*, 302
- Lenhossék, M. v. Ramon y Cajal's New Fibril Method, 639**
- Leon, Hyacinthe de.** *Myasthenic Paralysis*, 770
- Lepine, J.** *Tumour of Auditory Nerve*, 77
- Léri, André.** *Cécité et Tabes (Review)*, 721; *Retina in Tabetic Amaurosis*, 756; *Optic Nerve in Tabetic Amaurosis*, 813
- Levi and Malloizel.** *Hystero-Traumatism*, 155
- Levi, Hugo.** *Cervical Rihs*, 830
- Lewis, Thomas.** *Defective Development of Lateral Cerebellar Lobes in Dog*, 756
- Liepmann.** *Deep and Superficial Pain in Cerebral Hemiplegia*, 775
- Lloyd, J. H.** *Metaphysical Conception of Insanity*, 608
- Loeper and Crouzon.** *Hemiedema in Hemiplegia*, 777
- Loewenstein, H. Correl, and R. A. Hibbs.** *Congenital High Position of Scapula*, 558
- Loewy, E. and E. Berger.** *Secondary Contracture of Levator Palpebræ Superioris in Facial Paralysis*, 163
- Long, Ed.** *Cerebral Hemiplegia with Persistent Hemianæsthesia*, 600
- Low, G. C., and F. W. Mott.** *Tissues in Sleeping Sickness in European*, 593
- Lugaro, E.** *Pathology of Cells of Sensory Ganglia*, 228; *Hypochondriasis with Delusions of Persecution*, 477; *Unilateral Hallucinations of Hearing*, 608; *Neurone Theory*, 691
- Lugiato, Luigi.** *Ergographic Curve*, 223;



- Secondary Degenerations studied by Donaggio's Method, 811
- MAAS, O. A Rare Family Nervous Disease, 705
- M'Bride, C. A. Treatment of Inebriety by Atropine, 562
- M'Carthy, D. New Spino - Muscular Phenomenon, 164
- M'Kennan, T. M. T. Tumours of Brain, 462
- Macphail, Hinshelwood, and Ferguson. Word-blindness, with Right Homonymous Hemianopsia, 827
- Macpherson, John. Variation in its Relation to the Origin of Insanity, 285
- Malloizel and Levi. Hystero-Traumatism, 155
- Mann, Ludwig. Cerebellar Hemiataxia and its Origin, 661
- Mansfeld. Donath's Method of Demonstrating Presence of Choline in Cerebro-Spinal Fluid of Epileptics, 651
- Marburg, O. Basal Optic Root and Tractus Peduncularis Transversus, 386
- Marburg and Breuer. Apoplecticiform Bulbar Paralysis, 316
- Marchiafava and Bignami. Corpus Callosum in Alcoholism, 158
- Marie, Pierre, and Georges Guillain. Direct Pyramidal Tract and "Crescent" Pyramidal Tract, 56; Homolateral Pyramidal Tract, 56; Secondary Degenerations of Anterior Column of Cord, 693
- Marinesco. Fibrillar Part of Nerve Cells, 530; Neurofibrils, 749; Vibratory Sensibility, 774
- Marion. Craniectomy by Means of Gigli's Saw, 486
- Martin, A. Spinal Anæsthesia in Labour, 845
- May, W. Page. Innervation of Sphincters and Musculature of Stomach, 642
- Mazzei. Sero-therapy of Epilepsy, 482
- Media and Patini. Catatonia, 330
- Meige, Henri. Tic of the Eyes, 238; Facial Spasm, 78, 240
- Meige and Briessaud. Infantile Type of Giantism, 713
- Meige and Feindel. Tics of Childhood, 824
- Mellus, E. Lindon. A Nucleus Lateral to the Fasciculus Solitarius, 55
- Mendel, K. Reflex on Dorsum of Foot, 416; Special Flexor Reflex of Toes, 714
- Mercier and others. Criminal Responsibility, 837
- Merklen and Broc. Paralysis of Palate, 777
- Meyer, Adolf. Traumatic Insanity, 476
- Meyer, Dietetic Bromide Treatment of Epilepsy, 253
- Michotti, A. Nerve Fibres and their Bifurcation in Spinal Ganglia, 802
- Miécamp, L. Neurasthenic Headache, 607
- Milian. Nature of Tabes, 807.
- Mills, C. K. Cerebral Cortex of Man, 747; Aphasia and Cerebral Zone of Speech, 826
- Modena, G. Acromegaly, 466
- Montgomery, Alan, and L. Pierce Clark. Psychopathic Wards, Pavilions and Hospitals for American Cities, 780
- Montyel, Marandon de. The Voice in First and Second Stages of General Paralysis, 72; Predisposition in Etiology of Mental Diseases, 666
- Mooers, Emma W. Syphilis of Nervous System, 764
- Moor, L. de and D. de Buck. Morphology of Muscular Degeneration, 62
- Mosher, J. Montgomery. Albany Hospital Report, Department for Mental Diseases, 485
- Mosso. Respiratory Movements of Thorax and Diaphragm, 150; Fatigue (Review), 253
- Mott, F. W. Tremors, 776.
- Mott, F. W., W. D. Halliburton and Arthur Edmunds. Regeneration of Nerves, 586
- Mott, F. W., and G. C. Low. Tissues in Sleeping Sickness in European, 593
- Moynihan. Gastric Tetany, 159
- Mülbeyer. Family Amaurotic Idiocy, 250
- Munch. Surgical Treatment of Facial Paralysis, 334
- Murray, George B. Serum Treatment of Exophthalmic Goitre, 782
- Muskens, L. J. J. Pre-epileptic Stage, 766
- NAGEOTTE, J. Anatomical Study of Posterior Columns, 391; Radicular Lesions of Cord accompanying Cerebral Tumours, 591
- Neurath. Nervous Complications of Whooping-Cough, 772
- Newmark. Family Spastic Paraplegia, 762
- Nicholson, H. Oliphant. Thyroid Extract in Puerperal Eclampsia, 335
- Nisal. Lumbar Puncture in Psychiatry, 479
- OBERSTEINER. Arbeiten aus dem Neurologischen Institute an der Wiener Universität (Review), 336; Pigment in Nerve Cells, 389, 697; Pyramidal Variation in Mammals, 440

- Obici, Guilio. Action of Radium on Excitability of Cerebral Cortex, 396
- Okounoff. Involuntary Movements of Head as Expression of Affection of Semicircular Canal, 324
- Oppenheim, H. Certain Reflex Movements in Infantile Spastic Diplegia, 84; Myasthenia Gravis, 823
- PAGANO, G. Existence of Trophic Nerve, 220; Cerebellar Localisation, 585
- Pandy, K. Force of Tendon-Reflexes, 554
- Papadaki, A. Paralysis of Oculomotor Nerves in Tabes Dorsalis, 647
- Paris, A. and E. Rist. Cerebro-Spinal Meningitis due to Diplococcus of Weichselbaum, 311
- Parkinson, Porter. Post-basal Meningitis due to Pneumococcus Lanceolatus, 408
- Patini and Media. Catatonia, 830
- Pavy, F. W. Diabetic Neuritis, 645
- Pearce and Boston. Blood in Epilepsy, 535
- Peebles, A. M. S., and Lewis C. Bruce. Leucocyte Counts in Mental Disease, 666
- Pel, P. K. Myasthenia Pseudo-Paralytica, 771
- Perusini, G. Deaf-Mutism and Coën's Idiopathic Alalia, 471
- Pick, A. Compensatory Processes in Spinal Cord, 699
- Pic, A., and S. Bonnamour. Friedreich's Disease with Cerebral Softening, 541
- Pickett, William. Senile Dementia, 478
- Poli Cesare. Centrifugal Function of Posterior Roots of Spinal Cord, 453
- Pollack and Bielschowsky. Innervation of Mammalian Eye, 528
- Potts, C. G., and W. G. Spiller. Solitary Tubercle of Pons, 601
- Prout, Thomas P., and L. Pierce Clark. Status Epilepticus, 767
- Putnam, J. J. and J. W. Elliott. Tumour involving Spinal Cord, treated by Operation, 89
- QUAIFE, G. Methods for Preservation of Golgi Preparations, 219
- RABAND and Joffroy. Association of Tabes with General Paralysis of Insane, 71
- Rad, C. v. Tumours of Upper Cervical Cord and Medulla Oblongata, 707
- Raecke. Hysterical Insanity, 611
- Raimann, E. Hysterical Mental Affections (Review), 723
- Ransohoff. Emphysema of Skin after Epileptic Attack, 238
- Raymond, F. Parkinson's Disease, 465; Nervous Leprosy, 773
- Raymond and Cestan. Cirsoid Aneurism of Cervical Cord, 545
- Redlich, E. Association Systems of Mammalian Brains, 448
- Reich. Apparatus for Estimating Volume of Brain, 746
- Rennie, George E. Functional Astereognosis, 579; Myasthenia Gravis, 770
- Reynolds, Ernest S. Tabes Dorsalis, 177; Family Disseminated Sclerosis, 758
- Richartz. Tetany in Dilatation of Stomach, 655
- Rist, E. and A. Paris. Cerebro-Spinal Meningitis due to Diplococcus of Weichselbaum, 311
- Ritter, Gottfried von. Progressive Spinal Muscular Atrophy in Childhood, 315
- Roberts, W. F., and E. E. Southard. Chronic Internal Hydrocephalus in a Youth, 818
- Robertson, W. Ford. Method of producing Selective Deposit of Silver in Blocks of Tissue, 623; Pathology of Chronic Alcoholism, 403
- Roncoroni. Classification of Mental Diseases, 662
- Rüdin, E. Acute Hallucinatory Insanity in Prisoners, 87
- Rutkowski. Röntgen Rays in Neurology, 608
- Rydal. Hereditary Cerebellar Ataxy, 756
- SAINTON and Voisin. Trophædema, 778
- Sala, Guido. Finer Structure of Retina, 683
- Sand, René. Cortico-Bulbar and Cortico-Pontine Pyramidal Fibres, 441
- Sargent, Porter Edward. Optic Reflex Apparatus of Vertebrates, 748
- Scheiber, S. H. "Atypical" Tetany, 654
- Scheven. Physiology of Knee-Jerk, 587
- Schlesinger. Cytological Examination of Cerebro-Spinal Fluid, 651
- Schlittenhelm, A. Syringomyelia with Ocheiromegalia, 69
- Schuyten. Fatigue in School Children, 456
- Sciamaanna. Middle Finger Phenomenon, 324
- Seiffer. Paralysis of Spinal Accessory Nerve in Tabes Dorsalis, 67
- Senator, H. Aphasia with Left-sided Hemiplegia in Right-Handed Individuals, 553
- Serbaky. Dementia Præcox, 719
- Sherrington and Woodworth. A Pseud-

- Affective Reflex and its Spinal Path,** 695
- Smith, G. Elliot.** So-called "Affenspalte" in Human Brain, 55; "Limbus Postorbitalis" in Egyptian Brain, 142; Fossa Parieto-Occipitalis, 218; Pithecoïd Abnormality of Sylvian Region, 219; Communication between Musculo-Spiral and Ulnar Nerves, 221; An Aberrant Circumolivary Bundle Springing from the Left Pyramidal Tract, 377; "Transitory Fissures" of Human Brain, 442; Retro-Calcarine Region of Cortex Cerebri, 442; Morphology of Human Brain, 443; Morphology of Occipital Region of Cerebral Hemisphere, 443
- Sölder, von.** Corneo-mandibular Reflex, 163
- Soukhanoff, Serge.** Obsessions and Impulsions, 85; Intracellular Network of Golgi, 143; Endocellular Network in Nerve Elements of Spinal Ganglia, 390; Visceral Anæsthesias in General Paralysis, 470
- Soukhanoff and Boutenko.** Korsakoff's Disease, 245
- Soukhanoff, Geier, and Gourévitch.** Protoplasmic Processes of Nerve Cells, 802
- Soukhanoff and Wedensky.** Continuous Alcoholic Delirium, 325
- Southard, E. E. and W. F. Roberts.** Chronic Internal Hydrocephalus in a Youth, 313
- Spielmayer, W.** Symptomatology of Encephalitis, 599
- Spiller, W. G.** Muscular Atrophy caused by Encephalomyelitis, 34; Uncinate Group of Fits and Severe Subcutaneous Hæmorrhage in Epilepsy, 552
- Spiller and Camp.** Multiple Sclerosis, 649
- Spiller, W. G. and Charles H. Frazier.** Regeneration of Posterior Spinal Roots, 310
- Spiller, W. G. and C. S. Potts.** Solitary Tubercle of Pons, 601
- Spiller, W. G. and T. H. Weisenburg.** Partial Paralysis of Upper Limb from Vascular Lesion of Cord, 69; Sensory Segmental Zone of Umbilicus, 631
- Spitzer, A.** Relationship of Abnormal Bundles to Normal Brain Structure, 685
- Spitzer, A. and J. P. Karplus.** Abnormal Bundles in Human Brain-Stem, 684
- Squires.** Basilar Meningitis, 459
- Stanziale.** Syphilis and Progressive Paralysis, 336
- Starck.** Motor Function of Vagus, 807
- Starr, M. Allen.** Is Epilepsy a Functional Disease? 413
- Steinberg and Latzko.** Anencephaly, 152
- Steiner.** Cremasteric Reflex and Superposition of Reflexes, 555
- Stephenson, Sydney.** Congenital Word-Blindness, 711
- Stewart, Purves.** Diagnosis of Hysteria, 82; Unilateral Congenital Lesion of Medulla and Spinal Cord, 705
- Stoddart, W. H. B.** Evolution of Consciousness, 151
- Sträuber, Anton.** Histology of Multiple Sclerosis, 304
- Streeter, Hun and Blumer.** Myasthenia Gravis, 769
- Sullivan, W. O.** Social Causes of Alcoholism, 714
- Svenson, Frey.** Study of the Ego, 398
- Sym, William George.** Recurrent Paralysis of Third Nerve, 77
- TAYLOR, Alfred G.** Treatment of Chronic Internal Hydrocephalus, 783
- Taylor, James.** Subacute Combined Sclerosis, with Profound Anæmia, 648
- Thiellé.** Atrophy of Muscles of Thigh—Treatment by Sinusoidal Voltaisation, 831; Sciatica Treated by Oscillating Current, 844
- Thiemich.** Hysteria in Childhood, 159
- Thomas, André.** Treatment of Neurasthenia and Hysteria by Isolation and Psychotherapeutics, 668
- Thomas and Déjerine.** Verbal Blindness with Agraphia, 658; Sensory Aphasia, 711
- Thomas and Hauser.** Changes in Spinal Root Ganglia of Tabetics, 697; Pathological Histology and Pathogeny of Syringomyelia, 815
- Thomas, John Jenks, and P. C. Knapp.** Reflexes in Long Distance Runners, 297
- Thoyer - Rozat.** Obstetrical Paralysis, 757
- Toulouse and Vurpas.** Prolongation of Reaction of Pupils to Poisons in General Paralysis, 318
- Tredgold, A. F.** Disseminated Sclerosis, 497; Relations of Epilepsy to Amentia, 604
- Trepat.** Catatonic Dementia Præcox, 781
- Trevelyan, E. F.** "Some Observations on Tuberculosis of Nervous System," 64
- Treves, Z.** Nervous Fatigue and Energy of Contraction in Voluntary Muscular Work, 395; Myographic and Ergographic Curve, 590

- Turnbull, H. M. Bilateral Loss of Post-Central Cortex, 754
- Turner, John. Primary Staining of Rat's Brain by Methylene Blue, 639
- Turner, W. Aldren. Mental Condition in Epilepsy, 412; Salt Starvation in Treatment of Epilepsy, 793
- Turner, W. Aldren, and James Collier. Intra-Medullary Abscess of Spinal Cord, 819
- UGOLOTTI, F. Cerebral Sclerosis in Isolated Patches, 710
- Usher, C. H., and George Dean. Course of Optic Nerve Fibres, 386
- VARIOT. Partial Paralysis of Tongue and Lips in Girl aged 14, 236
- Verger, Henri, and Jean Abadie. Plantar Reflex, 415
- Voison and Sainton. Trophœdema, 778
- Voss. Tetany, along with Myotonic Symptoms, 658
- Vurpas and Toulouse. Prolongation of Reaction of Pupils to Poisons in General Paralysis, 318
- WALBRACH and Hall. Adiposis Dolorosa, 713
- Walton, G. L. Degeneracy and Degenerate, Plea for Supplementary Terms Deviation and Deviate, 252
- Warrington, W. B. Pneumococcal Meningitis, 107; Ventral Corneal Cells after Section of Posterior Roots, 311
- Waterston and Hepburn. Spinal Cord of Porpoise, 449
- Weber, F. Parke. Erythromelalgia, 468; Fœcal Vomiting in Functional Nervous Disease, 830
- Weber. Galloping Paralysis, 234
- Wedensky and Soukhanoff. Continuous Alcoholic Delirium, 325
- Weill and Gallavardin. Pathological Anatomy of Acute Diffuse Myelitis, 61
- Weisenburg, T. H. Triceps, Biceps, and Finger Clonus, 85; Uræmic Hemiplegia, 653
- Weisenburg, T. H. and W. G. Spiller. Partial Paralysis of Upper Limb from Vascular Lesion of Cord, 69; Sensory Segmental Zone of Umbilicus, 681; Injury to Lower Spinal Cord and Cauda Equina, 819
- Weismann, August. Heredity and Genetic Variation (Review), 847
- Westphal, A. Diagnostic Value of "Lidschlussreaction" of Pupils, 76
- Williamson, R. T. Hereditary Syphilitic Tabes, 435; Hemiplegia as Sign of Cerebral Tumour, 824
- Wilson, S. A. K. Eye Muscles in Organic Hemiplegia, 265; Choline in Cerebrospinal Fluid as sign of Nervous Tissue Degeneration, 651; Syringomyelia and Syringobulbia, 704
- Wilson, S. A. Kinnier, and O. Crouzon. Senile Combined Sclerosis of Spinal Cord, 431
- Wizel, Adam. Specific Delusions in General Paralysis, 332
- Woodworth and Sherrington. A Pseudo-Affective Reflex and its Spinal Path, 695
- Wright, Clarence A. Methods of Application of High Frequency Currents, 562
- Wright, Hamilton. Beri-beri in Monkeys, 409
- ZENNER, Philip. Alexia, 321
- Zimmermann, A. Electric Treatment of Facial Neuralgia, 843
- Zuckerkandl. Morphology of Ape's Brain, 442; Comparative Anatomy of Occipital Lobes, 442; Olfactory Radiations, 686; Collateral Sulcus, 746







FEB 15 1906



4101389

~~4101389~~  
~~4101389~~  
F

